PB-243 826

OCCUPATIONAL AND ENVIRONMENTAL PESTICIDE EXPOSURE STUDY IN SOUTH FLORIDA

John E. Davies

Miami University

Prepared for:

National Environmental Research Center

March 1975

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EPA-650/1-75-002

PB 243 826

March 1975

Environmental Health Effects Research Series

OCCUPATIONAL AND ENVIRONMENTAL PESTICIDE EXPOSURE STUDY IN SOUTH FLORIDA

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U.S. Environmental Protection Agency
Office of Research and Development
Washington, DC 20460

EPA-650/1-75-002

OCCUPATIONAL AND ENVIRONMENTAL PESTICIDE EXPOSURE STUDY IN SOUTH FLORIDA

by

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Contract No. 68-02-1277
ROAP No. 21AYL
Program Element No. 1EA078

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Prepared for

U. S. ENVIRONMENTAL PROTECTION AGENCY
OFFICE OF RESEARCH AND DEVELOPMENT
WASHINGTON, D. C. 20460

March 1975

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Publication No. EPA-650/1- 75-002

TECHNICAL REPORT DATA
(Please read Instructions on the reverse before completing)

1. REPORT NO.	2.	3. RECIPIENT'S ACCESSION NO. PB243826
4. TITLE AND SUBTITLE "OCCUPATIONAL & ENVIRONMENTAL PESTICIDE EXPOSURE STUDY IN SOUTH FLORIDA"		5. REPORT DATE 6. PERFORMING ORGANIZATION CODE
7. AUTHOR(S) John E. Davies, M.D.		8. PERFORMING ORGANIZATION REPORT NO.
9. PERFORMING ORGANIZATION NAME AND ADDRESS Department of Epidemiology & Public Health University of Miami School of Medicine P.O. Box 520875 Miami, Florida 33152		10. PROGRAM ELEMENT NO. 11. CONTRACT/GRANT NO. Contract No. 68-02-1277
12. SPONSORING AGENCY NAME AND ADDRESS U. S. Environmental Protection Agency Pesticides and Toxic Substances Effects Laboratory National Environmental Research Center Research Triangle Park, North Carolina 27711		13. TYPE OF REPORT AND PERIOD COVERED Annual 1/1/74-12/31/74 14. SPONSORING AGENCY CODE

15. SUPPLEMENTARY NOTES

16. ABSTRACT Multiresidue analysis of urinary pesticide metabolites offer an effective means of measurements of human exposure to the non-persistent pesticides. In studies of different degrees of human exposure to parathion DEP, a metabolite of paraoxon, was the most sensitive indicator of serious exposure. Concentrations in cases of >0.4 ug/ml were observed in first urines collected in 7 cases of poisoning with serious enzyme inhibition. In contrast, from 71 sequential urines in parathion exposed workers only 1 urine exceeded these concentration. The DEP:DETP ratio was equally informative; the mean ratio being 4.14 in 20 urines from the poison cases and 0.88 in urines of exposed workers, a difference which was significant at the <.01 level. Excretion of metabolites for 91 days after ingesting of Dichlofenthion was observed, emphasizing the significance of exposures to the less polar organophosphates in both acute and chronic effects. Low level exposures of 38 members of the general population showed that 29% were positive for Dursban exposure and 100% were positive for PCP, a frequency which suggested that this pesticide was as ubiquitous as DDT in man. A variety of pesticides were identified in a regular air monitoring program for pesticides in South Florida.

17. KEY WORDS AND DOCUMENT ANALYSIS

a. DESCRIPTORS	b. IDENTIFIERS/OPEN ENDED TERMS	c. COSATI Field/Group
1. Urinary metabolite surveillance of the occupational pesticide worker 2. Special significance of DEP the oxon metabolite in parathion exposure 3. Chronicity of human health effects of the less polar organophosphates. 4. Low level exposure to general population of non-persistent pesticides.		

18. DISTRIBUTION STATEMENT	19. SECURITY CLASS *(This Report)*	21. NO. OF PAGES 92
	20. SECURITY CLASS *(This page)*	22. PRICE 4.75 - 2.25

EPA Form 2220-1 (9-73)

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Abstract

In our Pesticide Epidemiological Field Studies in 1973 (EPA-650/1-74-009) we showed that the alkyl phosphates were highly significantly inversely correlated with red blood cell cholinesterase values, and this year our studies have confirmed that multiresidue analyses of urinary metabolites offer an effective measurement of human exposure to the non-persistent pesticides. Besides their epidemiologic potential, urinary metabolite information facilitated worker surveillance and simplified the diagnosis and management of acute pesticide poisoning.

In studies of different human exposures to parathion, DEP, the metabolite of paraoxon, proved to be a sensitive indicator of illness and serious cholinesterase inhibition. Thus, concentrations in excess of 0.4 ug/ml were observed in 7 parathion poisoning cases; yet, in 71 urines from 5 pesticide exposed workers having normal cholinesterase values, only 1 urine had concentrations of DEP >.4 ug/ml. The ratio of DEP:DETP concentrations was also very informative. The average ratio was 4.4 in 20 urines from the hospitalized cases but only 0.88 in the exposed workers, this difference was significant at the $p < .01$ level. The data suggested that in parathion exposure, if in a single urine, DEP concentrations were twice as high as DETP, and if the former were present in concentration of >.4 ug/ml, these were the hallmarks of over exposure. The data suggested that the whole problem of parathion intoxication could be ascribed to the amount of oxon present. In the re-entry situation, danger arises when paraoxon forms, and prevention will have to focus on the physical and chemical factors which reduce or prevent exogenous oxon formation. In applicator poisoning, illness appears to be the result from an over loading of the body with endogenously produced paraoxon. These urinary metabolite data appear to have a potential in validating re-entry times or in testing protective devices.

Prolonged metabolite excretion was encountered in a poisoning case which was the result of suicidal ingestion of dichlofenthion. Cholinesterase enzymes were 90% inhibited for 39 days and urinary metabolites were detected for 91 days. The clinical and toxicological features of this type of pesticide opens up a whole new dimension of pesticide toxicity because of the persistence of the effects. Expressions of low level exposures of the general population to the non-persistent pesticides was provided by urinary metabolite studies of 38 persons. All had levels of PCP and 29% were positive for 3,5,6-TC Pyridinol, a metabolite of Dursban.

Environmental studies were reflected by regular air monitoring of pesticides in several different sites of South Florida. Dichlofenthion, Dursban and Lindane were found in every urban sample, and Heptachlor and Alpha and Gamma Lindane and Dursban were also identified. Alpha-BHC, Diazinon, Lindane and Dursban were the pesticides most frequently identified in the Everglades. This activity has been one of the few ongoing air monitoring studies in the U.S. this last year and we were able to provide confirmatory evidence that as a result of the DDT ban no traces of this insecticide were found in the ambient air collected; the only exception being in a single sample from a pesticide formulating plant.

This report was submitted in fulfillment of Contract No. 68-02-1277 by the University of Miami School of Medicine under the sponsorship of the U.S. Environmental Protection Agency. Work was completed as of December 31st, 1974.

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ACKNOWLEDGEMENTS

The investigators acknowledge with gratitude the cooperation and assistance offered us to conduct these studies by Allied Helicopter Service Inc., Tri-State Dusting Service, South Bay Growers, U. S. Department of Agriculture, the Dade County Housing and Urban Development Program, the Eveready Pest Control Company, the Dade County Parks Department, Woodbury Chemical Company, and the several volunteers representative of the general population for their willing cooperation and participation to provide blood and urine samples for appropriate cholinesterase and urinary metabolite studies.

In addition, the investigators at the University of Miami appreciate the advice and guidance provided by Drs. Thomas Scotti and William Durham, Dr. Taalat M. Shafik, Dr. John Santolucito and Mr. Frank Wiliniski.

CONCLUSIONS

The goals of these studies have been directed toward a better understanding of different degrees of human pesticide exposure. When the pesticide worker is over exposed and starts to develop cholinergic manifestations, conventionally this exposure has been related to red blood cell and plasma cholinesterase. There are, however, several well recognized limitationswith these biological indices and certain urinary pesticide metabolites have shown to be more sensitive indices of exposure. The urinary alkyl phosphate metabolites were one group of pesticide metabolites whose potential as biological indices of pesticide exposure needed to be evaluated. In a study of these metabolites in a group of helicopter pilots and loaders having heavy work exposure to ethyl-methyl parathion mixtures, we found that there was excellent correlation of these metabolites with red cell cholinesterase levels. DEP, DETP, DMP and DMTP were all significantly inversely correlated.

Our studies this year have confirmed that both qualitative and quantitative information was provided by the alkyl phosphate data from parathion exposures. Concentrations of DEP were >.4 ug/ml in all the first urines in seven poisonings investigated. The entire urinary output during the first 48 hours of hospitalization were collected from three of the seven pesticide poisoning cases. In each specimen voided DEP concentrations were >.4 during this period. These werein striking contrast to the levels of DEP observed in asymptomatic occupationally exposed workers; only one out of 71 urines had a DEP concentration of >.4 ug/ml.

The DEP:DETP ratio was also highly informative; the mean of this ratio for these two metabolites was 4.14 in the poisoned cases compared to 0.88 in the 71 urines from the occupationally exposed group. These data clearly emphasized the qualitative significance of DEP. DEP is the alkyl phosphate derivative from ethyl paraoxon exposure which seems to be the only key factor in parathion poisoning. In reviewing all of the data we concluded that if in a single urine from a person having parathion exposure, DEP concentrations were twice as high as DETP, and if DEP concentrations were >.4 ug/ml these were the hallmarks of excessive exposure and were metabolite concentrations which were indicative of overt or imminent parathion poisoning.

In our experience this year multiresidue techniques have been found to be an excellent measure of human pesticide exposure to the biodegradable pesticides. On most occasions urinary alkyl phosphate data were complimentary to urine phenolic data, the combined information facilitating the identification of specific pesticide exposures.

In metabolite studies of workers exposed to pesticides where cholinesterase inhibition was neither observed nor anticipated, the chronicity of the metabolite excretion following a short work exposure was surprising. Alpha naphthol was detected 42 hours after a 5 hour exposure to carbaryl. Dursban metabolites were detectable for up to 30 hours after a 4½ hour exposure. Dichlofenthion (VC-13) produced the most protracted toxic effects of all pesticides studied. 90% inhibition of cholinesterase was observed for as long as 39 days after oral ingestion and both alkyl phosphate and phenolic metabolites could be detected for as long as 91 days after exposure. This concept of chronicity of organophosphate exposure could be attributed to the fat solubility of certain pesticides and the phenomenon raises a whole new dimension of acute and chronic effects. With compounds which are as fat soluble as dichlofenthion, if present use

continues it is not beyond the realm of possibility that these materials will be identified in the human adipose pesticide residue.

Both types of metabolites were also found to be of great value as measures of incidental exposure to the non-persistent pesticides. In a study of 38 members of the general population of Dade County every urine had traces of pentachlorophenol (PCP) and 29% were positive for 3,5,6-TC pyridinol (this is a metabolite of the compound Dursban). The concentrations of these metabolites in the general population were all of a low order of magnitude though PCP was as ubiquitous as DDT has been in the past.

The second goal of these studies has been to measure environmental air pollution by pesticides. The air monitoring data of pesticides presented in this report represents the only sources of pesticide concentrations in air in the United States last year. Significant qualitative and quantitative information was obtained. No DDT was identified in air samples except in a collection from a formulating plant. Dichlofenthion, Dursban and lindane were found in every sample from the suburban site and in the Everglades α- BHC, diazinon, lindane and dursban were the pesticides most frequently identified. Chlordane was identified for the first time as well as Heptachlor. In none of the sites were sizeable concentrations of pesticides detected with the exception of air levels in a formulating plant.

RECOMMENDATIONS

Our studies to date continue to reinforce the concept that both urinary alkyl phosphate and phenolic metabolites are excellent indices of human pesticide exposure. In our first year of research, they were shown to correlate with red cell cholinesterase levels, and in our studies this year, the special significance of the di-alkyl phosphate metabolite reflective of oxon exposure has been identified. More and more it appears that the presence of the oxon whether preformed exogenously on the leaves or endogenously as the result of saturating the body's detoxification processes is the cause of pesticide illness. It is recommended, therefore, that the potential of DEP and DMP in occupational exposure to the more toxic organophosphates, be further investigated through the acquisition of more data of these metabolites in acute and occupational exposures to parathion and Phosdrin. The relationship of these to cholinesterase inhibition needs to be validated.

The ratio of DEP:DETP has also been found to be very informative and the potential of a field test kit which can measure these two metabolites needs to be investigated. With the acquisition of data of this sort, it should be possible to measure the safety of the work environment. Metabolite concentrations should be expressed in micrograms per milliliter but these values should be corrected to a standard osmolality (800). Twelve hour urines, rather than each voided urine should be examined under both situations. The possibility of storage of the less polar organophosphates in fat should be investigated. Significant prolonged exposure effects appear to result from exposure to these more fat soluble pesticides and it is recommended that every attempt be made to determine whether these are beginning to be stored in human fat. The frequency of alkyl phosphates and phenols in the urine of the general population is conflicting and the need to resolve this issue is urgent.

We believe that there is a need to improve the gas chromatographic and detection systems in order to concentrate larger samples of urine including further cleanup; this is the only way that low level exposure of the United States population to the non-persistent pesticides can be monitored. In this respect, further epidemiological studies of pentachlorophenol (PCP) are needed. This metabolite is as ubiquitous as DDT yet it has never been extensively studied epidemiologically, and Shafik has recently identified residues of this pesticide in the adipose tissue of the general population.[18]

Expanded and representative air monitoring for pesticides is another area of monitoring where our studies seemed to have identified an unmet need.

SECTION III

SCOPE AND PURPOSE OF THE CONTRACT

The overall goals of this program seek to measure the occupational and environmental effects of pesticide exposure in South Florida. In addition to the conventional approaches of measuring exposures to the organophosphate and carbamates through cholinesterase determinations, special emphasis has been placed upon the study of exposure to these pesticides through the study of their urinary metabolites. The dialkyl phosphate and urinary metabolites have been examined under varying degrees of occupational, accidental and incidental exposure. In addition to the urinary metabolite studies possible central nervous system effects of occupational exposure have been examined through the collection of electroencephalographic tracings in workers. Environmental measurements of pesticide exposure have been quantitated on the basis of regular monitoring of ambient air samples in selected sites in South Florida.

This annual report describes the activities of the contract in these three areas and covers the contract period of January 1, 1974 through December 31, 1974. Much of these activities have already been reported in detail in the three preceeding quarterly reports. In order to avoid repetition the salient features of this year's study are described in detail, with special emphasis being given to research and toxicological findings.

SECTION IV

Background Information of Urinary Metabolite Studies - As a result of the Occupational Safety and Health Act of 1970 considerable emphasis has been focused on the introduction and enforcement of practices and standards which are designed to improve the safety and health of the worker in industry. The pesticide industry, and in particular the pesticide exposed worker, is an area of special concern. Appropriate groups in agriculture and health have responded to these challenges and collectively have developed programs related to the education, training, legislation and surveillance of the exposed worker. As a result of the switch from the more persistent organochlorine to the less persistent but more toxic organophosphate and carbamate insecticides, the need to control and contain worker exposure to these groups of pesticides has assumed increased importance.

From a practical point of view, worker exposure can be arbitrarily divided into those exposures which are associated, (1) with the manufacture, formulation, transportation and application of pesticides and (2) those exposures which are acquired during the processes of harvesting and thinning. In the former, the worker at some time or other, comes into contact with the concentrate; illness which results from such an exposure can be termed "applicator poisoning". In the latter type of exposure, sickness occurs as a result of contact with the pesticide residue on the leaf or in the soil of the work environment. This type of illness is sometimes referred to as "picker poisoning". The goals of industrial health in both situations are, firstly, to prevent acute poisoning, the symptoms and signs of which are cholinergic, and secondly, to prevent excessive and undesirable exposures, the chief manifestation of which is a biological effect rather than a clinical effect and is reflected by a decline of the red cell and plasma cholinesterase. Several preventive strategies for these two types of poisoning are currently in vogue. Picker poisoning, or residue intoxication involves a sizeable work force so that cholinesterase surveillance is impractical both on the grounds of worker acceptability and also from a purely logistical point of view. The federal government is currently in the process of developing re-entry times for this type of occupational illness. These times are prescribed intervals between application and picking which must lapse before agricultural workers enter the field for harvesting—different times for different pesticides are presently under review.

In the applicator situation, surveillance of the worker has traditionally relied upon cholinesterase testing; here however experience has shown that there are several serious setbacks with this approach. These include the necessity of obtaining pre-exposure levels and the absence of a definitive cholinesterase level which is diagnostic of overt illness. For these reasons, many workers in occupational health have explored the potential of the pesticide urinary metabolite as an alternative measure of human pesticide exposure, and as a result of new methodology significant progress has been made in the area, which suggest that these urinary indices of exposure could become effective expressions of worker exposure.

Metabolism studies have shown that for the biodegradable pesticides, the phenols, phenoxy acids and the alkyl phosphates are the major metabolites.

Before the advent of multiresidue analytical procedures, colorimetric techniques were used to study some of these metabolites. Paranitrophenol (PNP) was one of the earliest phenolic metabolites to be investigated and several researchers confirmed that PNP excretion was a more sensitive measure of parathion than blood cholinesterase levels.[1-3] Although these earlier approaches were able to detect levels of PNP as small as 0.01 ppm, large volumes of urine (100 ml) were required to detect these low levels and the method had additional complications which were the result of high background interferences. The potential for the study of low level human exposure to biodegradable pesticides by means of the halo and nitrophenol and phenoxy acids was greatly facilitated by the development of the multiresidue procedures (Shafik, Sullivan and Enos).[4] The principle behind these new approaches were based upon the preparation of the ethyl derivative of the phenols and acids. Similar multiresidue techniques were developed for low level exposures to the aromatic N methyl carbamate insecticides. Another approach emphasized the potential of alkyl phosphate metabolites during human pesticide exposure. Earlier methods relied upon the preparation of the ethyl and methyl ester derivatives of the various dialkyl phosphates and subsequent analysis by gas chromatography employing a phosphorus sensitive detector.[5-7] For both of these reasons the earlier techniques did not lend themselves to adaptation to field exposure studies on account of the length of the analytical procedure and because at low levels of dialkyl phosphate excretions interference from urinary inorganic phosphates were encountered. These earlier methods were modified by Shafik et al. in 1973, and their modified method permitted the study of the amyl derivatives of the dialkyl phosphates in human monitoring programs.[8] This modified technique involved the extraction of an acidified urine, derivitization with diazopentane, silica gel column chromatography and flame photometric gas chromatograph. Recoveries of the dialkyl phosphate metabolites from human urines spiked at 0.01 - 0.05 ppm were in the range of 80 - 100%. Only four alkyl phosphates were recognized in the non-exposed population and in a group of workers exposed to Dasanit, Thimet, and Di-Syston; these were the dimethyl phosphate (DMP), diethyl phosphate (DEP), dimethyl thioposhphate (DMTP), diethyl thiophosphate (DETP). Limits of detectability were 0.01 ppm for the dialkyl phosphates and 0.02 for the diethyl phosphorothionates. In six samples from 6 individuals with no record of exposure to these non-persistent pesticides, the mean concentration of DMP was 0.012 ppm, for DEP it was 0.05 ppm, for DMTP it was 0.06 ppm and DETP was detected in only one of the six specimens. In the 6 samples of the occupationally exposed, the respective means for these metabolites were DMP 0.02 ppm, DEP 0.97 ppm, DMTP 0.09 ppm and DETP 0.60 ppm. Thus, DEP and DETP concentrations were much higher in the exposed group than the general population.

In our project during 1973 we applied this modified method to the study of different occupational exposure groups working in the field. These groups included five highly exposed aircraft loaders and four intermediately exposed pilots and three non-exposed persons. The major exposure of these workers was to ethyl-methyl parathion (6-3) mixtures. In the highest exposed group, the average DMP was 0.25 ppm, the average DEP was 0.48 ppm, the average DMTP was 0.09 ppm and the average DETP was 0.49 ppm. In the intermediate group, the averages for these respective metabolites were 0.06 ppm, 0.26 ppm, Not Detected and 0.11 ppm respectively. None of the alkyl phosphates were detected in the three non-exposed persons. Since these exposures were predominately due to the more toxic pesticides severe red blood cell and plasma cholinesterase inhibition were frequently encountered in the more exposed group. It was thus

possible to correlate concentrations of these metabolites with cholinesterase levels. DEP, DETP, and DMP were significantly inversely correlated with red blood cell (RBC) and plasma (PL) cholinesterase (ChE) levels; positive correlations were observed with PNP levels. All were significant at the "P" <.001 level. Thus, our studies confirmed that the urinary metabolites were an excellent measurement of occupational exposure and might possibly be used to predict cholinesterase inhibition and illness if the qualitative and quantitative characteristics of the metabolies were known. We do not know at what concentrations and which of the alkyl phosphates were the most important when occupational exposures were becoming excessive. Additional studies were clearly necessary in workers who were occupationally exposed to the more toxic pesticides. In this area, parathion mixtures and phosdrin are known to be the major offenders in worker sickness. The hazard from these more toxic pesticides were the exposure situations which needed much further study.

Apart from being probable predictors of cholinesterase inhibition and illness, preliminary data on the excretion of the urinary metabolites were also obtained following exposure to the less toxic pesticides. Here cholinesterase inhibition was not to be expected nor was it encountered. What was observed, however, was an unexpectedly prolonged excretion of the alkyl phosphate and phenolic metabolites. It soon became obvious therefore that the study of these metabolites also had considerable epidemiologic potential.

Urinary metabolite studies following single exposure made us realized that several of these organophosphates particularly the pesticides containing halo phenol moieties were soluble in fat, and this was an area which certainly needed further investigation. Another area of uncertainty was in the descriptive characteristics of these metabolites under conditions of incidental exposure. Very little was known of the magnitude and distribution of these pesticides in the population at large and information was needed as to how they were produced and how could the data be interpreted.

These were some of the unknowns recognized as a result of our first year's activities. In addition, grab samples of urine had been obtained and nothing was known about the sequential excretion of these urinary metabolites. The studies described in this report take over from this point. In the area of urinary metabolites we have attempted to throw some light in several areas and have studied urinary metabolites under differing occupational pesticide exposure situations and after exposure to several different pesticides. The alkyl phosphate metabolites that were encountered in several pesticides are listed in Table 1 and the material and methods, results and discussion of the several pesticide exposure studies are presented separately with reference to each of the individual pesticides studied.

Description of Phases

The studies this year were not conducted in predetermined chonological order and the only planned exposure study was a special dichlofenthion (VC-13) investigation. Cholinesterase determinations were made at different times in various exposure groups; these were used to justify different exposure categories. Sequential urinary metabolite data were collected during and after single and multiple pesticide exposures. Additional data was collected from the general population and from individuals hospitalized as a result of over exposure to the pesticide under study.

Table 1. LIST OF PESTICIDES DISCUSSED IN THIS REPORT AND THEIR DIALKYL PHOSPHATE AND PHENOLIC METABOLITES

Pesticide	Metabolite
A. Dialkyl Phosphates	
Ethyl parathion	DETP, DEP
Methyl parathion	DMTP, DMP
VC-13	DETP, DEP
DDVP	DMP
Cygon (Dimethoate)	DMTP, DMP, DMDTP
Phosdrin (Mevinphos)	DMP
Thimet (Phorate)	DETP, DEP, DEDTP
Dursban	DETP, DEP
B. Phenols	
Ethyl parathion	Paranitrophenol (PNP)
Methyl parathion	Paranitrophenol (PNP)
Dichlofenthion (VC-13(R))	2,4-Dichlorophenol (2,4-DCP)
Dursban	3,5,6-TC Pyridinol (3,5,6-TCP)
Pentachlorophenol	PCP
2,4-D	Free phenoxy acid
2,4,5-T	Free phenoxy acid
Silvex	Free phenoxy acid
Ronnel	2,4,5-Trichlorophenol (2,4,5-TCP)
Gardona	2,4,5-Trichlorophenol (2,4,5-TCP)
Lindane	2,4,5-Trichlorophenol (2,4,5-TCP)
	2,3,4,6 Tetrachlorophenol
	2,3,4,5 Tetrachlorophenol
	2,4,6 Trichlorophenol
	2,3,5 Trichlorophenol
Sevin	Alpha naphthol

Phase I - Cholinesterase categorization of different exposure groups.

Phase II - Urinary pesticide metabolite studies during and after ethyl-methyl parathion exposures.

Phase III - Urinary pesticide metabolite studies during and after work exposure to Dursban and DDVP.

Phase IV - Urinary pesticide metabolite studies during and after phorate (Thimet) exposure.

Phase V - Urinary pesticide metabolite studies during and after carbaryl (Sevin) exposure.

Phase VI - Urinary metabolite studies following acute and occupational exposure to dichlofenthion (VC-13).

Phase VII - Urinary pesticide metabolite studies in the general population.

Cholinesterase Categorization of Different Pesticide Exposure Groups

Background Information - Determination of the red blood cell and plasma cholinesterase enzyme level is the traditional method of measuring relative degrees of human exposure to the organophosphate and carbamate insecticides. Pesticides workers particularly the applicator groups, tend to have lower than average levels of both these enzymes especially if the occupational exposure has been to the more toxic organophosphate pesticides. In our studies of occupational effects in South Florida it was obviously necessary therefore to categorize the different occupational work groups under study with respect to average levels of these cholinesterase enzymes.

Materials and Results - The Michel method was used in all determinations.[9] Table 2 presents the man and ranges of these enzymes in several differing work exposure categories. As might be expected, the six hospitalized cases were the most severely exposed group and the average RBC and plasma cholinesterase (ChE) in this group was 0.36 and 0.19 ΔpH/hr respectively. The mean of the RBC ChE values was slightly greater than plasma ChE levels because post-PAM bloods were included in this group and reactivation effects are being reflected in the RBC ChE mean. The pesticide loaders, applicators (pilots) and formulators were the next most highly exposed group and the physical nature of their work and the opportunities for contact and exposure to parathion and Phosdrin were the major factors in the exposure of this group. The mean RBC ChE was 0.59 ΔpH/hr and the mean for plasma ChE was 0.57 ΔpH/hr. The intermediate exposure group consisted of agricultural and park sprayers as well as structural pest control operators. The mean RBC ChE was 0.77 ΔpH/hr and the mean plasma was 0.75 ΔpH/hr. The average plasma cholinesterase levels of structural pest contorl operators (SPCO) (0.53 ΔpH/hr) was lower in the two groups because of their predominant use of Dursban. The low exposure group were made up of agricultural inspectors and parks groundkeepers and the average levels of both enzymes from this exposed group were no different than from the averages of the general population.

Several individuals were subject to repeated cholinesterase determinations, either for reasons of surveillance and worker protection, or because of various

Table 2. MEAN AND RANGES OF RED BLOOD CELL AND PLASMA CHOLINESTERASE (ΔpH/HR) OF DIFFERENT PESTICIDE EXPOSURE GROUPS. 1974.

Group (number)	No. of Specimens	RBC ChE (ΔpH/hr)/ mean	RBC ChE (ΔpH/hr)/ ranges	Plasma ChE (ΔpH/hr)/ mean	Plasma ChE (ΔpH/hr)/ ranges
A. HIGH EXPOSED GROUP					
Acute Exposure - Hospitalized Cases (6)	6	0.36	0.08 - 0.55//	0.19	0.07 - 0.50//
Occupational Group					
Pilots (6)	22	0.55	0.26 - 0.75	0.67	0.30 - 1.02
Loaders (6)	8	0.52	0.25 - 0.80	0.56	0.30 - 0.90
Formulators (9)	34	0.67	0.20 - 0.90	0.56	0.35 - 1.09
Sub Total (21)	64	0.61	0.20 - 0.90	0.60	0.30 - 1.09
ALL HIGH EXPOSED GROUP (27)	70	0.59	0.08 - 0.90	0.57	0.07 - 1.09
B. MEDIUM EXPOSED GROUP					
Park Sprayers (3)	6	0.91	0.68 - 1.15	0.78	0.52 - 1.05
Agricultural Sprayers (14)	14	0.70	0.50 - 0.90	0.87	0.51 - 1.44
Structural Pest Control Operators (7)	9	0.77	0.68 - 0.90	0.53	0.20 - 0.77
ALL MEDIUM EXPOSED GROUP (24)	29	0.77	0.50 - 1.15	0.75	0.20 - 1.44
C. LOW EXPOSED GROUP					
US Dept. of Agriculture Inspectors (26)	26	0.64	0.60 - 1.20	0.81	0.60 - 1.20
Parks Groundkeepers (53)	74	0.73	0.41 - 1.30	0.88	0.55 - 0.91
ALL LOW EXPOSED GROUP (69)	100	0.71	0.50 - 0.91	0.84	0.46 - 1.30
D. GENERAL POPULATION (13)	13	0.74	0.60 - 0.89	0.83	0.65 - 1.10

/Michel method
//Post PAM blood

various project activities such as electroencephalographs or urinary metabolite studies. The results of these are shown in Table 3. The only serious RBC ChE decline was seen in case #8 whose RBC ChE exhibited a progressive decline between 2/25/74 and 5/3/74. He was taken off flying as a result of these levels and earlier urinary metabolite studies of DEP concentrations and DEP:DETP ratios were diagnostic of undesirable levels of parathion exposure.

Urinary Metabolite Studies Following Acute and Occupational Exposure to Ethyl-Methyl Parathion (6-3)

Materials and Methods - Sequential urines were collected after dermal exposures to 6-3 ethyl methyl parathion concentrations in two occupationally exposed workers and in one 4½ year old child. The rate of excretion of the urinary alkyl phosphate and paranitrophenol pesticide metabolites during the period of recovery in these three symptomatic cases were compared to the rate of excretion of these same metabolites in four asymptomatic pesticide workers during and for a period of up to 48 hours after being exposed to a 6-3 ethyl methyl parathion mixture singly or in combination with Phosdrin, Cygon or parathion 8E. The occupationally exposed workers included an aircraft loader and three pilots. The loader (W.G.) first mixed and then loaded parathion 6-3 mixtures into a fixed wing aircraft. He wore a long sleeve shirt and gloves while mixing and loading. The three pilots were helicopter aerial applicators and were employees of Allied Helicopter Service Inc. They did not mix or load the chemical themselves and during application each wore coveralls, a crash helmet and respirator, gloves and rubber boots. They were partially enclosed in a plastic bubble when flying. When not flying they would remove their protective clothing and spend time in the office adjoining the helicopter hanger. The drums of the pesticide concentrate were stored outside the hanger.

From the three symptomatic cases of acute exposure, each voided urine was collected during the period of hospitalization by the nursing staffs of the two hospitals involved. From the asymptomatic occupationally exposed groups urines were collected under the supervision of the field investigator during the day and by the volunteer himself during his time at home after receiving proper instructions. Urines were collected from the pilots during and after a period of 48 hours after pesticide application, during which time the pilots were sitting around the hanger, waiting for the weather to clear and during which time they were supposedly not occupationally exposed. Two separate series of urines were collected from the loader one month apart; on both occasions he was mixing and loading 6-3 ethyl methyl parathion and urines were collected during work exposure and for a period of 48 hours after. During the second exposure study he also mixed and loaded Cygon and Sevin, and in the post exposure period he was either at home or sitting around the work site.

Sequential urine samples were collected in hexane washed jars. The volume and time of voiding were recorded with each specimen. The specimens were stored in dry ice for up to four days and shipped to Miami where they were frozen at -15° and later analyzed for phenolic and alkyl phosphate metabolites.

The Michel method was used for RBC and plasma ChE and the Shafik et al. modification for the gas liquid chromatography analysis of alkyl phosphate metabolites in urine.[9,8] The Shafik et al procedure was used for the halogen and nitrophenols in urine and the Shafik et al procedure for alpha naphthol in urine.[10,11]

Table 3. SEQUENTIAL RED CELL AND PLASMA CHOLINESTERASE[#] LEVELS (ΔpH/hr) IN DIFFERENT EXPOSURE GROUPS. SOUTH FLORIDA. JANUARY - DECEMBER 1974.

Name	Occupational Group	Date		Date		Date		Date		Date		Date		Mean	
		RBC ChE	Pl. ChE	RBC ChE	Pl. ChE	RBC ChE	Pl. ChE	RBC ChE	Pl. ChE	RBC ChE	Pl. ChE	RBC ChE	Pl. ChE	RBC ChE	Pl. ChE
		1/29/74				3/29/74		6/7/74				10/28/74			
1. John P.	Formulator	0.78	0.47	3/14/74		0.65	0.45	0.85	0.60	8/14/74		0.70	0.45	0.75	0.50
2. Archie P.	"	0.53	0.40	0.73	0.35	0.70	0.40	0.60	0.54	0.82	0.45	0.76	0.40	0.69	0.42
3. Tommy W.	"	0.75	0.40	0.70	0.20	0.70	0.35	0.90	0.35	0.89	0.58	0.60	0.35	0.76	0.37
4. Steve P.	Marginally Exposed at Formulating Plt	0.70	0.72	0.70	0.45	0.77	0.60			0.72	0.90	0.72	0.60	0.72	0.53
5. Lewis T.	"	0.50	0.74	0.50	0.43	0.58	0.50			0.60	0.60	0.63	0.52	0.56	0.56
6. Francis B.	"	0.72	0.70	0.68	0.50			0.45	0.78	0.80	0.80			0.61	0.70
		2/25/74		3/9/74		4/22/74		4/27/74		11/11/74					
7. Frank B.	Helicopter Pilot	0.45	0.68	0.50	0.73	0.30	0.30	0.30	0.30	0.69	0.65			0.45	0.51
				3/14/74				5/3/74							
8. Danny A.	" "	0.46	0.69	0.41	0.68	0.26	0.50	0.26	0.50					0.35	0.59
				4/22/74		11/11/74									
9. Gordon J.	" "	0.59	0.70	0.59	0.70	0.73	1.02							0.64	0.81
		4/22/74		7/24/74											
10. Dick D.	" "	0.57	0.54	0.70	0.50	0.63	0.75								
		1/16/74		3/27/74		5/29/74		6/13/74		10/3/74					
11. Robert S. Parks	Sprayman	1.00	0.60	1.06	0.52	0.88	0.50	1.02	0.69	1.15	0.65			1.02	0.59
12. Estan O.	" "	0.81	1.05	0.68	1.03	0.68	0.60	0.80	1.15					0.74	0.96

[#]Michel method

Results - Exposure Data of Symptomatic Cases - The exposure histories of the three persons acutely ill revealed that all were the results of excessive exposure to parathion mixtures. T.B., was a 59 y/o w/m employed as a mixer in a fixed wing aircraft company. On 10/8/74 he started working at 6:00 a.m. loading an aircraft with parathion 6-3, Lannate, and a 30% methyl parathion and Toxaphene mixture. He wore rubber gloves but was otherwise unprotected. Within one hour he began to develop cholinergic symptoms and was hospitalized by 11:30 a.m. Large amounts of atropine and IV infusions of 2-PAM were necessary for his recovery, and his post-PAM blood showed an RBC and plasma ChE level of 0.55 ΔpH/hr and 0.55 ΔpH/hr respectively.

J.D., a 17 y/o w/m was also an aircraft loader and he too developed cholinergic symptoms after mixing and loading ethyl-methyl parathion (6-3) and Lannate. He was hospitalized at 7:00 p.m. on 11/17/74 and received large amounts of 2-PAM. The RBC and plasma ChE levels were 0.15 pH/hr and 0.10 pH/hr respectively. M.P., the third case was a 4½ y/o w/m who at 2:00 p.m. 5/19/74 while playing in a barn accidently spilled 1½ ounces of ethyl parathion concentrate on his blue jeans which he wore until 6:00 p.m. By 8:30 p.m. he was in acute cholinergic distress and was hospitalized shortly thereafter. The post-PAM cholinesterase levels of RBC and Pl. ChE were 0.55 ΔpH/hr and 0.18 ΔpH/hr respectively. Large amounts of paranitrophenol were identified in all three cases providing confirmatory evidence of parathion exposures.

Exposure Data of Asymptomatic Pesticide Workers - The exposure histories and cholinesterase levels of the loader and three pilots were presented in Table 4. All were in good health during the study period.

Sequential Urinary Pesticide Data of Symptomatic Cases - Tables 5, 6 and 7 present the individual concentrations of the alkyl phosphate metabolites and PNP levels from three individuals during their period of hospitalization on account of cholinergic manifestations.

Sequential Urinary Pesticide Data of Asymptomatic Cases of Pesticide Workers - Tables 8, 9, 10, 11 and 12 present similar data from the symptomatically exposed workers. Although all had occupational exposures to parathion mixtures, the occupationally exposed workers had additional work exposures to Phosdrin, Cygon and Sevin; the first two would add additional dimethyl alkyl phosphate metabolites. Exposures to ethyl parathion would be reflected by the excretion of the diethyl thiophosphates (DETP) and PNP, and the oxidation of the intact compound to paraoxon would result in the excretion of diethyl phosphate (DEP) and PNP. Exposure to methyl parathion would result in the appearance of dimethyl thiophosphate (DMTP) and PNP in the urine and the oxidation of the intact compound to paraoxon would result in the excretion of dimethyl phosphate (DMP) and PNP. In addition, Phosdrin would elicit a DMP response and Cygon would result in DMP, DMTP, DMDTP, although the latter metabolite is not encountered in occupational types of exposure. Sevin exposure would be recognized by the excretion of alpha naphthol. Because of the mixed exposure that some of the workers encountered, the only urinary metabolites that were specifically reflective of exposure to ethyl-methyl parathion 6-3 were the diethyl metabolites of ethyl parathion (DETP and DEP). The excretion of these metabolites therefore was especially studied in this comparison of 6-3 ethyl-methyl parathion exposures. Ethyl parathion metabolizes to the oxon which is known to be far more toxic than is the parent compound so that DEP reflects a greater potential for toxicity than does DETP.

Table 4. EXPOSURE HISTORY AND CHOLINESTERASE LEVELS (ΔpH/hr) OF OCCUPATIONALLY EXPOSED ASYMPTOMATIC PESTICIDE WORKERS.

Name	Category	Date	Chemical	Time	Exposure time in minutes	Cholinesterase† RBC	Cholinesterase† Plasma
W.G.	Loader	1/26/74	Parathion 6 - 3	9:15 - 11:15 am	120	0.60	0.80
W.G.	Loader	2/27/74	Parathion 6 - 3	9:00 - 11:30 am	150		
		2/28/74	Sevin	7:30 - 11:30 am	240		
		2/28/74	Cygon	1:00 - 6:30 pm	330		
P.B.	Pilot	2/24/74	Parathion 6 - 3	4:00 - 4:10 pm	10	0.45	0.68
			Parathion 6 - 3 and Phosdrin	4:30 - 8:25 pm	235		
		2/25/74	Parathion 6 - 3	8:45 - 9:00 am	15		
			Parathion 8E	5:25 - 7:05 pm	100		
G.J.	Pilot	2/24/74	Phosdrin	10:05 - 10:55 am	50	0.59	0.70
			Cygon	5:00 - 5:05 pm	5		
			Parathion 6 - 3	6:55 - 7:35 pm	40		
D.A.	Pilot	2/24/74	Parathion 6 - 3	10:05 - 10:20 pm	15	0.46	0.69
			Parathion 8E and Cygon	10:30 - 11:30 am	60		

† Michel method (ΔpH/hr)

Table 5. SEQUENTIAL URINARY EXCRETION RATES OF ALKYL PHOSPHATES AND PARANITROPHENOL IN A SPRAYMAN (T.B.) HOSPITALIZED FOR ACUTE DERMAL PARATHION INTOXICATION.

	Date	Time	ml voided	DMP ug/ml	DMP ug/vol voided	DMP ug/hour	DMTP ug/ml	DMTP ug/vol voided	DMTP ug/hour
Hospital Admission ##	10/08/74	11:35 am							
Initial urine collection	10/08/74	5:00 pm-11:00 pm	500	0.348	174.0	29.0	0.259	129.5	21.6
	10/08-09/74	11:00 pm- 5:00 am	315	0.256	80.6	13.4	0.119	37.5	6.2
	10/09/74	5:00 am-11:00 am	250	0.083	20.8	3.5	0.069	17.3	2.9
	10/09/74	11:00 am- 5:00 pm	800	0.045	36.0	6.0	ND	ND	ND
	10/10/74	5:00 am-11:00 am	250	0.194	48.5	8.1	0.091	22.8	3.8
	10/11/74	? 8:00 pm	210	0.025	5.3	?	ND	ND	ND
	10/11/74	8:00 pm- 8:45 pm	165	ND	ND	ND	ND	ND	ND
	10/11/74	8:45 pm- 9:15 pm	165	ND	ND	ND	ND	ND	ND
	10/11/74	9:15 pm-10:00 pm	210	ND	ND	ND	ND	ND	ND
	10/11/74	10:00 pm-11:30 pm	330	ND	ND	ND	ND	ND	ND
	10/11-12/74	11:30 pm-12:50 am	275	ND	ND	ND	ND	ND	ND
	10/12/74	12:50 am- 1:30 am	400	ND	ND	ND	ND	ND	ND
	10/12/74	1:30 am- 2:45 am	800	ND	ND	ND	ND	ND	ND
	10/12/74	2:45 am- 4:15 am	200	ND	ND	ND	ND	ND	ND
	10/12/74	4:15 am- 4:45 am	250	ND	ND	ND	ND	ND	ND
	10/12/74	4:45 am- 6:15 am	400	ND	ND	ND	ND	ND	ND
	10/12/74	6:15 am- 6:45 am	625	ND	ND	ND	ND	ND	ND

##Post-PAM cholinesterase RBC 0.55 ΔpH/hr and plasma 0.55 ΔpH/hr

Methyl parathion and ethyl parathion identified in blood at a level of 3 ppb and 14.3 ppb respectively

ND=Not detectable;

Limits of detectability: DMP 0.015, DMTP 0.026

?=Previous hour not known

Table 5 (Continued)

	Date	Time	ml voided	DEP ug/ml	DEP ug/vol voided	DEP ug/hour	DETP ug/ml	DETP ug/vol voided	DETP ug/hour	PNP ug/ml	PNP ug/vol voided	PNP ug/hour
Hospital admission##	10/08/74	11:35 am										
Initial urine collection	10/08/74	5:00 pm-11:00 pm	500	0.882	441.0	73.5	0.330	165.0	17.5	2.14	1,070.	170.0
	10/8-9/74	11:00 pm- 5:00 am	513	0.672	211.7	35.3	0.252	74.4	13.2	0.880	277.	46.2
	10/09/74	5:00 am-11:00 am	250	0.257	64.3	10.7	0.167	41.8	7.0	0.630	157.	26.3
	10/09/74	11:00 am- 5:00 pm	800	0.183	146.4	24.4	ND	ND	ND	0.204	163.	27.34
	10/10/74	5:00 am-11:00 am	250	0.595	148.8	24.8	0.061	15.3	15.3	0.590	147.	24.6
	10/11/74	? - 8:00 pm	210	0.102	21.4	?	0.073	15.3	?	0.186	39.1	?
	10/11/74	8:00 pm- 8:45 pm	165	0.044	7.3	UC	ND	ND	ND	0.064	10.6	UC
	10/11/74	8:45 pm- 9:15 pm	165	0.031	5.1	UC	ND	ND	ND	0.030	5.0	UC
	10/11/74	9:15 pm-10:00 pm	210	0.025	5.3	UC	ND	ND	ND	0.048	5.8	UC
	10/11/74	10:00 pm-11:30 pm	330	0.018	5.9	4.0	ND	ND	ND	0.048	15.8	10.6
	10/11-12/74	11:30 pm-12:50 am	275	0.042	11.6	8.7	ND	ND	ND	0.064	17.6	13.2
	10/12/74	12:50 am- 1:30 am	400	0.064	25.6	UC	ND	ND	ND	0.052	20.8	UC
	10/12/74	1:30 am- 2:45 am	800	0.028	22.4	17.9	ND	ND	ND			
	10/12/74	2:45 am- 4:15 am	200	0.039	7.8	5.2	ND	ND	ND			
	10/12/74	4:15 am- 4:45 am	250	0.059	14.75	UC	ND	ND	ND	0.078	19.5	UC
	10/12/74	4:45 am- 6:15 am	400	ND	ND	ND	ND	ND	ND			
	10/12/74	6:15 am- 6:45 am	625	0.056	35.0	UC	ND	ND	ND	0.64	40.0	UC

ND=Not detectable
Limits of detectability: DEP 0.020; DETP 0.032
?=Previous hour not known
UC=Unable to calculate less than 1 hour

Table 6. SEQUENTIAL URINARY EXCRETION RATES OF ALKYL PHOSPHATES AND PARANITROPHENOL IN A SPRAYMAN (JD) HOSPITALIZED FOR ACUTE PARATHION INTOXICATION - DERMAL EXPOSURE

A. ALKYL PHOSPHATE DATA

	Date	Time	ml void	DMP			DMTP			DEP			DETP		
				ug/ml	ug/vv	ug/hr	ug/ml	ug/vv	ug/hr	ug/ml	ug/vv	ug/hr	ug/ml	ug/vv	ug/hr
Hospital Admission##	11/19/74	7:00 pm													
Initial Urine	11/20/74	7:15 am-4:50 pm	276	0.372	102.7	10.7	0.038	10.48	1.1	0.992	273.8	28.6	0.096	26.5	2.8
	11/20-21/74	4:50 pm-8:30 am	554	0.504	279.2	17.8	0.096	53.2	3.4	0.278	711.3	45.4	0.278	154.0	9.8
	11/21/74	8:30 am-10:19 am	186	0.235	43.7	24.1	0.029	5.39	3.0	0.824	153.3	84.4	0.090	16.7	9.2
		10:19 am-12:30 pm	288	0.336	96.8	42.4	0.081	23.3	10.2	0.896	258.1	113.0	0.195	56.2	24.6
		12:30 pm-8:40 pm	69	0.284	19.6	2.4	0.039	2.69	0.3	0.796	54.9	6.7	0.080	5.52	0.64
	11/21-22/74	8:40 pm-5:30 am	358	0.388	138.9	15.7	0.057	20.4	2.3	0.972	347.98	39.4	0.174	62.3	7.1
	11/23/74	12:47 am-8:00 am	292	0.222	64.8	8.98	0.040	11.7	1.6	0.676	197.39	27.4	0.12	35.0	4.9

B. PHENOLIC DATA

	Date	Time	ml void	PNP		
				ug/ml	ug/vv	ug/hr
Initial Urine	11/20/74	7:15 am-4:50 pm	276	1.14	314.6	32.8
	11/20-21/74	4:50 pm-8:30 am	554	2.36	1307.4	83.5
	11/21/74	8:30 am-10:19 am	186	0.88	163.7	90.1
		10:19 am-12:30 pm	288	1.4	403.2	176.6
		12:30 pm-8:40 pm	69	0.6	41.4	5.1
	11/21-22/74	8:40 pm-5:30 am	358	0.92	329.4	37.3
	11/23/74	12:47 am-8:00 am	292	0.64	186.9	25.9

RBC ChE 0.15 ΔpH/hr and Pl. ChE 0.10 ΔpH/hr
Methyl parathion 3.3 ppb and ethyl parathion 14.3 ppb in blood

Table 7. URINARY ALKYLPHOSPHATE AND PHENOLIC LEVELS IN AN ACUTE INTOXICATION OF PARATHION IN A 4½ YEAR OLD WHITE MALE (MATTHEW P.), DADE COUNTY, FLA.

	Date	Hour	ml	DMP			DMTP			DEP			DETP			PNP		
				ug ml	ug vv	ug hr	ug ml	ug vv	ug hr	ug ml	ug vv	ug hr	ug ml	ug vv	ug hr	ug ml	ug vv	ug hr
Hospital Admission##	5/19/74	9:30 pm																
Initial Urine	5/20/74	7 am-1 pm	200	0.298	59.6	9.9	0.342	68.4	11.4	2.21	442.0	73.7	1.48	296.0	49.3	2.26	452.0	75.3
	5/21/74	1 am-7am	439	0.067	29.4	4.9	0.342	150.1	25.	0.456	200.2	33.4	0.177	77.7	13	0.5	219.5	36.5
		7 am-1 pm	550	0.042	23.1	3.9	0.304	167.2	27.8	0.265	145.8	24.3	0.127	69.9	11.6	0.186	102.3	17.1
		1 pm-7 pm	320	0.045	14.4	2.4	0.351	112.3	18.7	0.252	80.6	13.4	0.108	34.6	5.8	0.206	65.9	10.9
	5/21-22/74	7 pm-1 am	283	0.042	11.9	2.	0.364	103.0	17.2	0.211	59.7	10.	0.094	26.6	4.4	0.198	56.0	9.3
	5/22/74	1 am-7 am	650	ND	ND	ND	0.649	421.9	70.	0.123	80.0	13.3	0.128	83.2	13.9	0.134	87.1	14.5
		7 am-8 am	40	ND	ND	ND	0.577	23.1	23.1	0.091	3.6	3.6	0.101	4.0	4.	0.086	3.4	3.4
	6/16/74	7:15-8:10 pm	15	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND

##Post-Pam RBC ChE 0.55 ΔpH/hr and Pl. ChE 0.18 ΔpH/hr
ND=Not Detectable
Limits of detectability; DMP 0.03; DMTP, DEP and DETP 0.04
PNP 0.009

Table 8. SEQUENTIAL ALKYL PHOSPHATE AND PARANITROPHENOL EXCRETIONS IN A LOADER AFTER TWO HOURS EXPOSURE TO MIXING AND LOADING ETHYL METHYL PARATHION. DADE COUNTY. 1974 (W.G. #1).

Dade 1974	Hour	ml voided	DMP			DMTP			DEP			DETP			PNP		
			ug/ml	ug/vol voided	ug/hr.	ug/ml	ug/vol voided	ug/hr.	ug/ml	ug/vol voided	ug/hr.	ug/ml	ug/vol voided	ug/hr	ug/ml	ug/vol voided	ug/hr.
1/26	9:45 am	225	ND	ND	ND	0.145	32.6	26.1	ND	ND	ND	0.061	13.7	11.0	0.025	5.6	4.5
	11:30 am	250	ND	ND	ND	0.118	29.5	16.9	ND	ND	ND	0.093	23.3	13.3	0.034	8.5	4.9
	2:30 pm	295	ND	ND	ND	0.079	23.3	7.8	ND	ND	ND	0.192	56.6	18.9	0.064	18.9	6.3
	7:15 pm	400	ND	ND	ND	0.193	77.2	16.3	ND	ND	ND	0.116	46.4	9.8	0.21	84.0	17.7
1/27	1:00 am	400	0.056	22.4	3.9	0.155	62.0	10.8	0.058	23.2	4.0	0.084	33.6	5.8	0.187	74.8	13.0
	6:35 am	475	0.069	32.8	5.9	0.087	41.3	7.4	0.058	27.6	4.9	0.184	87.4	15.7	0.124	58.9	10.5
	10:43 am	475	0.063	29.9	7.2	0.070	33.3	8.0	0.061	29.0	7.0	0.297	141.1	34.1	0.09	42.8	10.3
	6:20 pm	425	0.053	22.5	3.0	0.077	32.7	4.3	0.062	26.4	3.5	0.254	108.0	14.2	0.108	45.9	6.0
	10:00 pm	395	0.026	10.3	2.8	0.074	29.2	8.0	0.029	11.5	3.1	0.204	80.6	22.0	0.039	15.4	4.2
1/28	2:30 am	425	0.042	17.9	4.0	0.082	34.9	7.8	0.047	20.0	4.4	0.183	77.8	17.3	0.059	25.1	5.6
	7:15 am	400	ND	ND	ND	0.090	36.0	5.3	ND	ND	ND	0.170	68.0	10.1	0.032	12.8	1.8

ND=Not detectable

Limits of detectability are: DMP 0.02, DEP 0.02, DMTP 0.03 and DETP 0.03 for alkyl phosphates
PNP 0.009 for phenols

Table 9. SEQUENTIAL ALKYL PHOSPHATE AND PARANITROPHENOL EXCRETIONS IN A LOADER (W.G. #2) AFTER 2½ HOURS EXPOSURE TO MIXING AND LOADING 6-3 ETHYL METHYL PARATHION AND 12 HOURS MIXING AND LOADING CYGON AND SEVIN. DADE COUNTY 1974

A. Alkyl Phosphate Data

Date 1974	Hour	ml voided	DMP ug/ml	DMP ug/vol voided	DMP ug/hour	DMTP ug/ml	DMTP ug/vol voided	DMTP ug/hour	DEP ug/ml	DEP ug/vol voided	DEP ug/hour	DETP ug/ml	DETP ug/vol voided	DETP ug/hour
2/27	11:30 am	300	ND	ND	ND	0.174	52.2	13.10	0.044	13.2	3.3	0.065	19.5	4.9
	2:00 pm	250	ND	ND	ND	0.102	25.5	10.20	0.039	9.8	3.9	0.123	30.8	12.3
	9:00 pm	250	0.042	10.5	1.5	0.059	14.8	2.10	0.071	17.8	2.5	0.141	35.3	5.0
	11:15 pm	200	0.035	7.0	3.1	0.107	21.4	9.50	0.052	10.4	4.6	0.080	16.0	7.1
2/28	1:30 am	200	0.049	9.8	4.4	0.089	17.8	7.90	0.078	15.6	6.9	0.067	13.4	6.0
	4:30 am	400	0.026	10.4	3.5	0.163	65.2	21.70	0.042	16.8	5.6	0.071	28.4	9.5
	7:00 am	250	0.022	5.5	2.2	0.156	39.0	15.60	0.034	8.5	3.4	0.074	18.5	7.4
	5:30 pm	175	0.029	5.1	0.5	0.049	8.6	0.82	0.061	10.7	1.0	0.097	17.0	1.6
	6:30 pm	225	0.031	7.0	7.0	0.070	15.8	15.80	0.072	16.2	16.2	0.070	15.8	15.8
3/1	1:30 am	250	0.038	9.5	1.4	0.059	14.8	2.10	0.055	13.8	2.0	0.130	32.5	4.6
	3:20 am	340	0.025	8.5	4.6	0.061	20.7	11.30	0.051	17.3	9.5	0.109	37.1	20.2
	7:08 am	340	0.033	11.2	3.0	0.043	13.6	3.60	0.058	19.7	5.2	0.155	52.7	13.9
	9:00 am	250	0.028	7.0	3.8	0.048	12.0	6.40	0.056	14.0	7.5	0.102	25.5	13.7

B. Phenolic Data

Date 1974	Hour	ml voided	PNP ug/ml	PNP ug/vol voided	PNP ug/hour	Alpha-Naphthol ug/ml	Alpha-Naphthol ug/vol voided	Alpha-Naphthol ug/hour
2/27	11:30 am	300	0.220	66.0	17.5			
	2:00 pm	250	0.262	65.5	26.2			
	9:00 pm	250	0.428	107.0	5.3			
	11:15 pm	200	0.406	81.2	36.1			
2/28	1:30 am	200	0.270	54.0	24.0			
	4:30 am	400	0.226	90.4	30.1			
	7:00 am	250	0.118	29.5	11.8	0.0073	1.83	0.73
	5:30 pm	175	0.218	38.2	3.6	0.0220	3.9	0.40
	6:30 pm	225	0.203	45.7	45.7	0.014	3.2	3.2
3/1	1:30 am	250	0.263	65.8	9.4	0.0273	6.8	1.0
	3:20 am	340	0.110	37.4	20.4	0.0173	5.9	3.2
	7:08 am	340	0.105	35.7	9.4	0.0155	5.3	1.4
	9:00 am	250	0.073	18.3	9.8	0.0302	7.6	4.1

ND=Not detectable
Limits of detectability for alkyl phosphates are: DMP 0.02, DEP 0.02, DMTP 0.03 and DETP 0.04 and for phenols they are: PNP 0.009 and alpha naphthol 0.006

Table 10. URINARY ALKYL PHOSPHATE EXCRETIONS IN A HELICOPTER PILOT (F.B.) DURING AND AFTER OCCUPATIONAL EXPOSURE TO PARATHION MIXTURES, PHOSDRIN AND CYGON. SOUTH FLORIDA 1974

Date 1974	Hour	ml voided	DMP			DMTP			DEP			DETP			PNP		
			ug/ml	ug/vol voided	ug/hour	ug/ml	ug/vol voided	ug/hour	ug/ml	ug/vol voided	ug/hour	ug/ml	ug/vol voided	ug/hour	ug/ml	ug/vol voided	ug/hour
2/24	3:15 pm	325	ND	ND	UC	0.287	93.28	UC	0.034	11.05	UC	0.098	31.85	UC	0.052	16.9	UC
	5:20 pm	100	0.047	4.7	2.26	0.195	19.50	9.36	0.111	11.1	5.33	0.320	32.00	15.4	ND	ND	ND
2/25	11:20 am	175	0.023	4.03	UC	0.137	23.98	UC	0.030	5.25	UC	0.03	5.25	UC	0.026	4.6	UC
	12:30 pm	175	0.035	6.13	6.13	0.155	27.13	27.13	0.078	13.65	13.65	0.045	7.88	7.8	0.048	8.4	8.4
	7:20 pm	175	ND	ND	ND	0.131	22.93	3.36	0.086	15.25	2.2	0.063	11.03	1.6	0.042	7.4	1.1
	8:45 pm	125	0.030	3.75	2.65	0.443	55.38	39.1	0.088	11.00	7.77	0.172	21.50	15.2	0.130	16.3	11.5
2/26	6:10 am	175	0.090	15.75	UC	0.169	29.58	UC	0.262	45.85	UC	0.441	77.16	UC	0.022	3.9	UC
	8:30 am	100	0.080	8.0	3.4	0.132	13.20	5.7	0.197	19.7	8.44	0.372	37.20	15.9	0.048	4.8	2.1
	9.43 am	200	0.042	8.4	6.9	0.352	70.40	57.9	0.055	11.0	9.04	0.113	22.60	18.6	0.080	16.0	13.2
	11:00 am	425	ND	ND	ND	0.262	111.35	86.8	0.037	15.72	12.55	0.065	27.63	21.5	0.038	16.2	12.6
	4:10 pm	150	0.063	9.45	1.83	0.199	29.85	5.8	0.187	28.05	5.43	0.350	52.50	10.2	0.02	0.02	0.02
	6:30 pm	150	0.037	5.55	2.38	0.081	12.15	5.2	0.085	12.75	5.46	0.251	37.65	16.1	0.02	0.02	0.02
	7:30 pm	150	0.028	4.2	4.2	0.115	17.25	17.25	0.054	8.1	8.1	0.127	19.05	19.0	0.096	14.4	14.4
	9:50 pm	150	0.022	3.3	1.41	0.318	47.70	20.44	0.044	6.6	2.83	0.175	26.25	11.2	0.144	21.6	9.3
2/27	6:00 am	325	0.035	11.38	1.4	0.107	34.78	4.26	0.111	36.08	4.4	0.279	90.68	11.1	0.030	9.8	1.2
	7:30 am	50	0.049	2.45	1.63	0.084	4.20	2.80	0.137	6.85	4.57	0.152	7.60	5.1	0.088	4.4	2.9
	9:00 am	125	0.042	5.25	3.5	0.105	13.13	8.75	0.105	13.13	8.75	0.150	18.75	12.5	0.018	2.3	1.5
	9:50 am	150	ND	ND	ND	0.059	8.85	8.85	0.048	7.42	7.2	0.141	21.15	21.1	0.062	9.3	9.3
	10:30 am	250	ND	ND	ND	0.056	14.0	14.0	ND	ND	ND	0.087	21.75	21.7	0.022	5.5	5.5

ND=Notdetectable

Limits of detectability: DMP 0.02 DEP 0.02, DETP 0.03, DMTP 0.03 and PNP 0.009

UC=Unable to calculate previous hour not known

Table 11. URINARY ALKYL PHOSPHATE AND PARANITROPHENOL EXCRETION IN A HELICOPTER PILOT (G.J.) 24 HOURS AFTER OCCUPATIONAL EXPOSURE TO PARATHION, PHOSDRIN AND CYGON. 1974

Date 1974	Hour	ml voided	DMP ug/ml	DMP ug/vol voided	DMP ug/hour	DMTP ug/ml	DMTP ug/vol voided	DMTP ug/hour	DEP ug/ml	DEP ug/vol voided	DEP ug/hour	DETP ug/ml	DETP ug/vol voided	DETP ug/hour	PNP ug/ml	PNP ug/vol voided	PNP ug/hour
2/25	11:45 am	300	0.050	15.0	10.6	0.231	69.3	48.9	0.129	38.7	27.3	0.058	17.4	12.3	0.160	48.0	33.9
	3:00 pm	108	0.051	5.5	1.7	0.059	6.37	2.0	0.180	19.4	6.0	0.127	13.4	4.2	0.252	27.2	8.4
	5:05 pm	225	0.051	11.5	5.5	0.153	34.4	16.5	0.128	49.1	23.5	0.145	32.6	32.6	0.338	76.1	36.5
	7:50 pm	525	ND	ND	ND	0.119	62.5	22.7	0.069	36.2	13.2	ND	ND	ND	0.096	50.4	18.3
	10:35 pm	225	0.039	8.78	3.2	0.151	34.0	12.4	0.116	26.1	9.5	0.113	25.4	9.3	0.274	61.7	22.4
2/26	5:30 am	275	0.070	19.3	2.8	0.120	33.0	4.8	0.172	47.3	6.8	0.165	45.4	6.6	0.336	92.4	13.4
	7:05 am	441	ND	ND	ND	0.109	48.1	30.4	ND	ND	ND	0.065	28.7	18.1	0.060	26.5	16.7
	8:50 am	320	ND	ND	ND	0.107	34.2	19.6	ND	ND	ND	0.055	17.6	10.1	0.032	10.2	5.9
	10:34 am	225	0.039	8.78	5.1	0.028	6.30	3.6	0.193	43.4	25.1	0.079	17.8	10.3	0.336	75.6	43.6
	1:00 pm	225	ND	ND	ND	ND	ND	ND	0.137	30.8	12.7	0.031	6.97	2.9	0.168	37.8	15.5
	3:25 pm	201	ND	ND	ND	ND	ND	ND	0.106	21.3	8.8	0.037	7.43	3.0	0.190	38.2	15.8
	5:25 pm	358	ND	ND	ND	ND	ND	ND	0.061	21.8	10.9	ND	ND	ND	0.072	25.8	12.9
	8:20 pm	226	ND	ND	ND	ND	ND	ND	0.048	10.9	3.7	ND	ND	ND	0.076	17.2	5.9
2/27	2:15 am	215	ND	ND	ND	ND	ND	ND	0.120	30.7	5.2	0.088	22.5	3.8	0.308	78.8	13.3
	4:50 am	325	ND	ND	ND	ND	ND	ND	0.069	22.4	8.9	0.06	18.5	7.0	0.094	30.6	11.8
	7:30 am	225	0.035	7.87	2.9	ND	ND	ND	0.097	21.8	8.2	0.05	10.6	3.9	0.132	29.7	11.1
	9:00 am	340	ND	ND	ND	ND	ND	ND	0.060	20.4	13.6	ND	ND	ND	0.050	17.0	11.3

ND=Not detectable
Limits of detectability: DMP 0.03, DEP 0.04, DMTP 0.03 and DETP 0.03
PNP 0.009

Table 12. URINARY ALKYL PHOSPHATE AND PARANITROPHENOL EXCRETION RATES IN A HELICOPTER PILOT (DA) 24 HOURS AFTER OCCUPATIONAL EXPOSURE TO PARATHION MIXTURES AND CYGON.

Date 1974	Hour	ml Voided	DMP ug/ml	DMP ug/vol voided	DMP ug/ hour	DMTP ug/ml	DMTP ug/vol voided	DMTP ug/ hour	DEP ug/ml	DEP ug/vol voided	DEP ug/ hour	DETP ug/ml	DETP ug/vol voided	DETP ug/ hour	PNP ug/ml	PNP ug/vol voided	PNP ug/ hour
2/25	11:05 am	160	0.083	13.28	UC	0.032	53.1	UC	0.240	38.4	UC	0.171	27.36	UC	0.092	39.6	UC
	4:00 pm	250	0.128	32.0	6.5	0.317	79.25	16.1	0.446	111.5	22.7	0.309	77.25	15.7	0.540	135.0	27.5
	7:30 pm	150	0.067	10.05	2.3	0.342	51.3	14.7	0.199	29.85	8.5	0.185	27.75	7.9	0.282	42.3	12.1
	8:48 pm	220	0.044	9.68	7.5	0.345	75.9	58.4	0.134	29.48	22.7	0.140	30.8	23.7	0.172	37.8	29.1
2/26	7:20 am	60	0.085	5.1	UC	0.198	11.9	UC	0.395	23.7	UC	0.284	17.0	UC	0.470	28.2	UC
	8:55 am	275	0.035	9.63	6.1	0.244	67.1	42.4	0.169	46.48	29.4	0.146	40.15	25.4	0.112	30.8	19.5
	10:35 am	350	0.029	10.15	6.1	0.273	95.55	57.3	0.081	28.35	17.0	0.127	44.45	26.7	0.046	16.1	9.7
	3:45 pm	200	0.050	10.0	1.9	0.285	57.0	11.0	0.211	42.2	8.2	0.220	44.0	8.5	0.236	47.2	9.1
	5:25 pm	160	0.056	8.96	5.4	0.294	47.0	28.2	0.246	39.36	23.6	0.249	39.84	23.9	0.340	54.4	32.6
	6:30 pm	125	0.073	9.13	8.4	0.210	26.25	24.2	0.343	42.88	39.6	0.284	35.5	32.8	0.364	45.5	42.0
2/27	7:30 am	75	0.059	4.43	UC	0.320	24.0	UC	0.377	28.28	UC	0.214	16.1	UC	0.408	30.6	UC
	10:25 am	260	0.033	8.58	2.9	0.218	56.68	19.4	0.142	36.92	12.7	0.137	35.62	12.2	0.102	26.5	9.2

UC=Unable to calculate previous hour not known

The excretion levels of these several metabolites are shown in Table 5 through 12. The data are expressed in three ways, namely, micrograms per milliliter (ug/ml), micrograms per volume voided (ug/vv) and micrograms excreted per hour (ug/hr). We have come to realize that expressions of data such as micrograms excreted per hour are subject to considerable error, especially if the collections are made under field conditions. The volumes of urines excreted at each voiding are subject to inaccuracies as is also exact time of voiding. Too often, it has been our experience, that participants tend to approximate to the nearest 1/2 hour rather than record the exact time of voiding. In addition, since part of the sample collection is during the leisure hours at home there is no certainty that the total 24 hour output is being accurately collected. The micrograms of the metabolite excreted per hour are calculated by multiplying the micrograms of the metabolite per milliliter of urine by the volume of urine voided and dividing this by the time interval between each voiding. Thus, two series of errors are built into the quantitation of these exposures of these metabolites when expressed in micrograms per hour.

With this principle in mind therefore the following figures 1 through 8 compare the excretion of these metabolites in the three symptomatic individuals during their period of hospitalization with the profile observed in the occupationally exposed group. Pesticide metabolite concentrations were expressed in micrograms per milliliter (ug/ml) in all these samples.

It will be apparent that both quantitative and qualitative differences exist in the alkyl phosphate and PNP excretion rates when the symptomatic groups are compared with the asymptomatic occupational group. The first point that is suggested by this comparison is a quantitative one. In all first urines, concentrations of DEP were >0.4 ug/ml; concentrations of this metabolite were at or above this level in all of the urines voided within the first 48 hours of hospitalization. In contrast, in the 71 urines analyzed from the asymptomatic group only one sample was >0.4 ug/ml. When we reviewed four other cases of ethyl methyl parathion intoxications not described in this report we were able to confirm these findings, and Table 13 lists the concentrations of DEP, DETP and PNP in the first urines collected and cholinesterase levels in seven cases of parathion poisonings. It will be noted that all had DEP concentrations of >0.4 ug/ml and all were dermally over exposed except for case #3 who ingested parathion, and was therefore an oral exposure rather than a dermal exposure; highest levels of DEP were seen in this case.

The second point that is suggested from this comparison is a qualitative one. In all three of the parathion poisoning cases, in all the urines collected during 48 hours of hospitalization, concentrations of DEP were always greater than the concentrations of DETP. The same was true for the additional poisoning cases listed in Table 13. If the relationship of these two metabolites to each other is expressed as a ratio, the DEP:DETP ratio was considerably greater than unity. In the five occupational studies, the picture was reversed in all but two individuals (G.J. and D.A.), and DETP concentrations were for the most part greater than DEP concentrations. It is difficult to explain why this was not the case with the two exceptions but it will be noted that the differences between DEP and DETP in these individuals were not nearly so striking as were the differences of these two metabolites in the

Figure 1. SEQUENTIAL URINARY EXCRETION RATES OF ALKYL PHOSPHATES AND PARANITROPHENOL IN A SPRAYMAN (TB) HOSPITALIZED FOR ACUTE DERMAL PARATHION EXPOSURE.

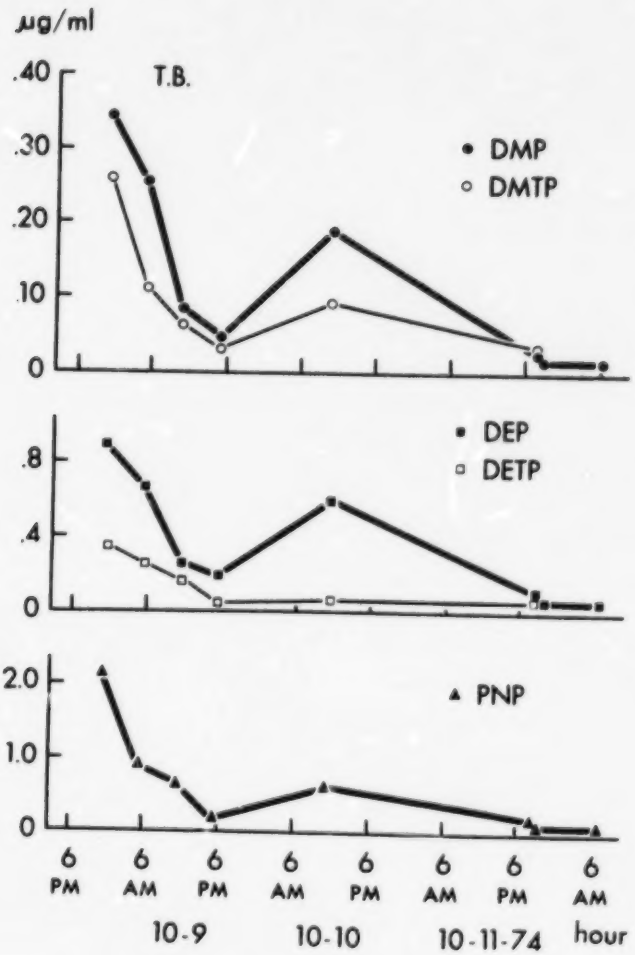


Figure 2. SEQUENTIAL URINARY EXCRETION RATES OF ALKYL PHOSPHATES AND PARANITROPHENOL IN A SPRAYMAN (JD) HOSPITALIZED FOR ACUTE PARATHION INTOXICATION - DERMAL

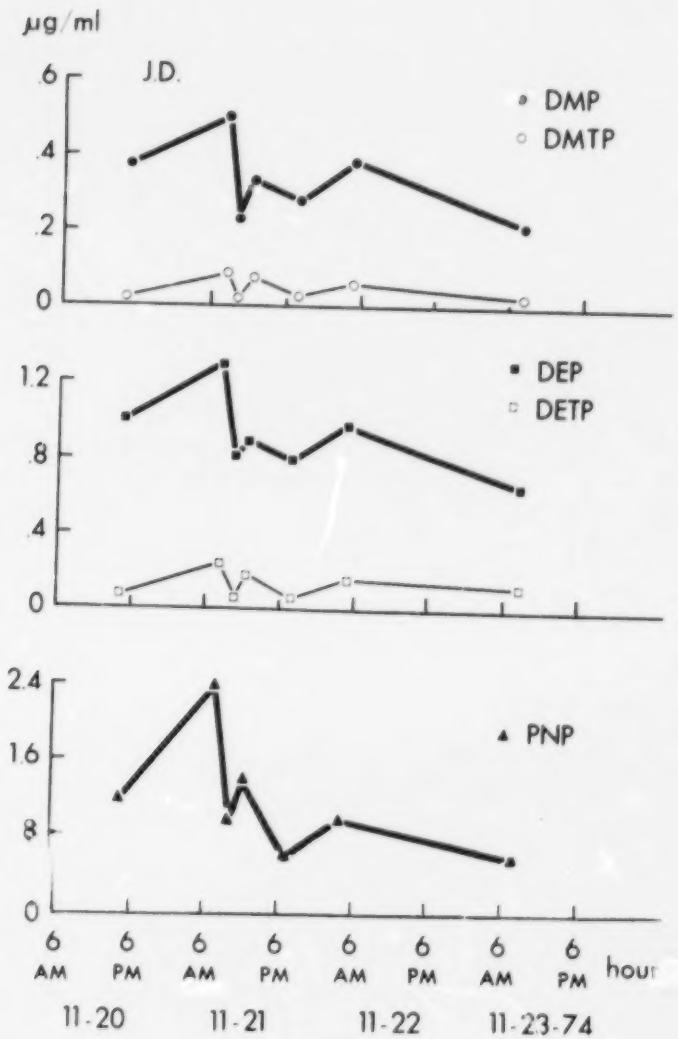


Figure 3. SEQUENTIAL URINARY EXCRETION OF ALKYL PHOSPHATE AND PARANITROPHENOL IN A 4 1/2 YEAR OLD WHITE MALE (MP) HOSPITALIZED FOR ACUTE PARATHION INTOXICATION - DERMAL

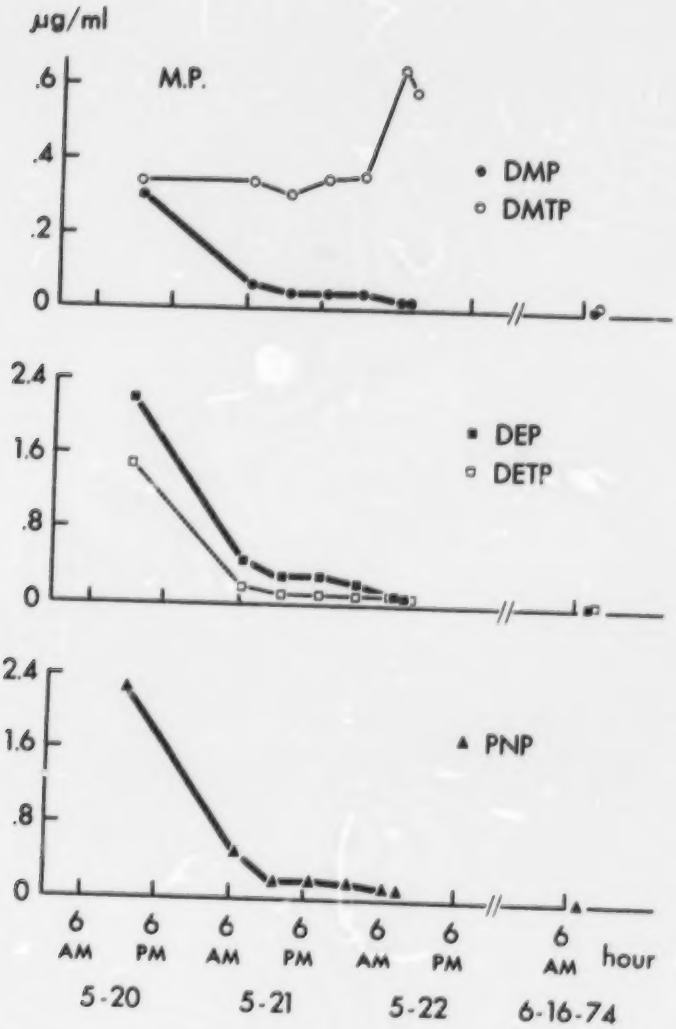


Figure 4. SEQUENTIAL ALKYL PHOSPHATE AND PARANITROPHENOL EXCRETIONS IN A LOADER AFTER TWO HOURS EXPOSURE TO MIXING AND LOADING 6-3 ETHYL METHYL PARATHION - DADE COUNTY, 1974 (WG #1)

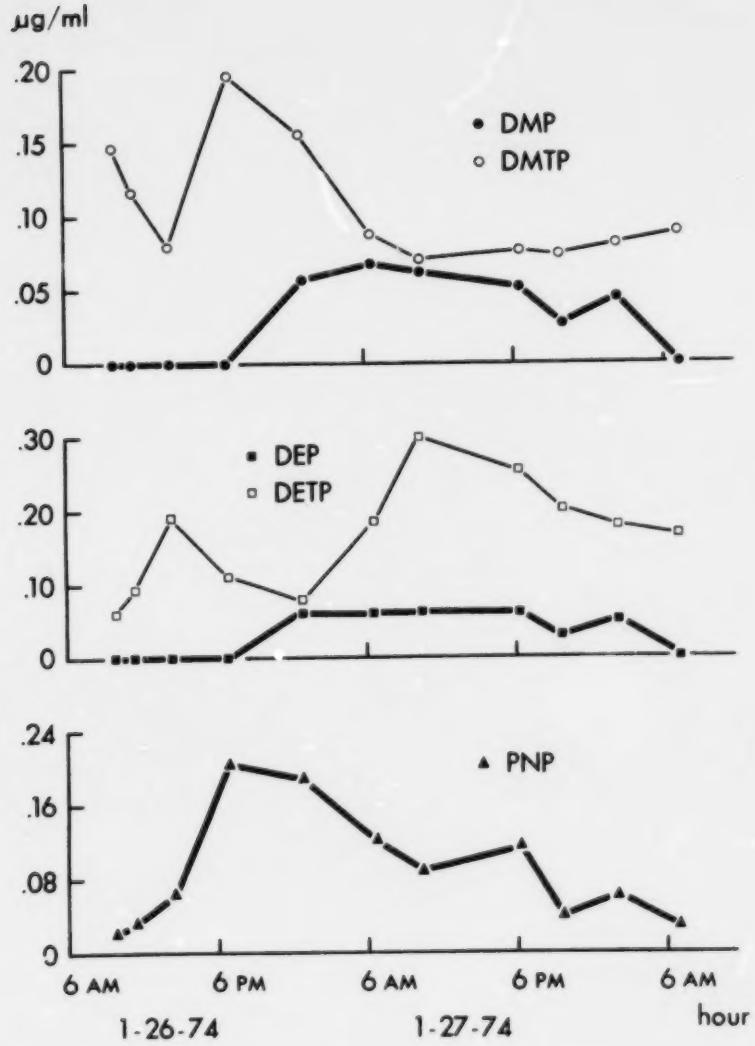


Figure 5. SEQUENTIAL ALKYL PHOSPHATE AND PARANITROPHENOL EXCRETIONS IN A LOADER (WG #2) AFTER 2 1/2 HOURS EXPOSURE TO MIXING AND LOADING 6-3 ETHYL METHYL PARATHION AND 12 HOURS MIXING AND LOADING CYGON DADE COUNTY, 1974

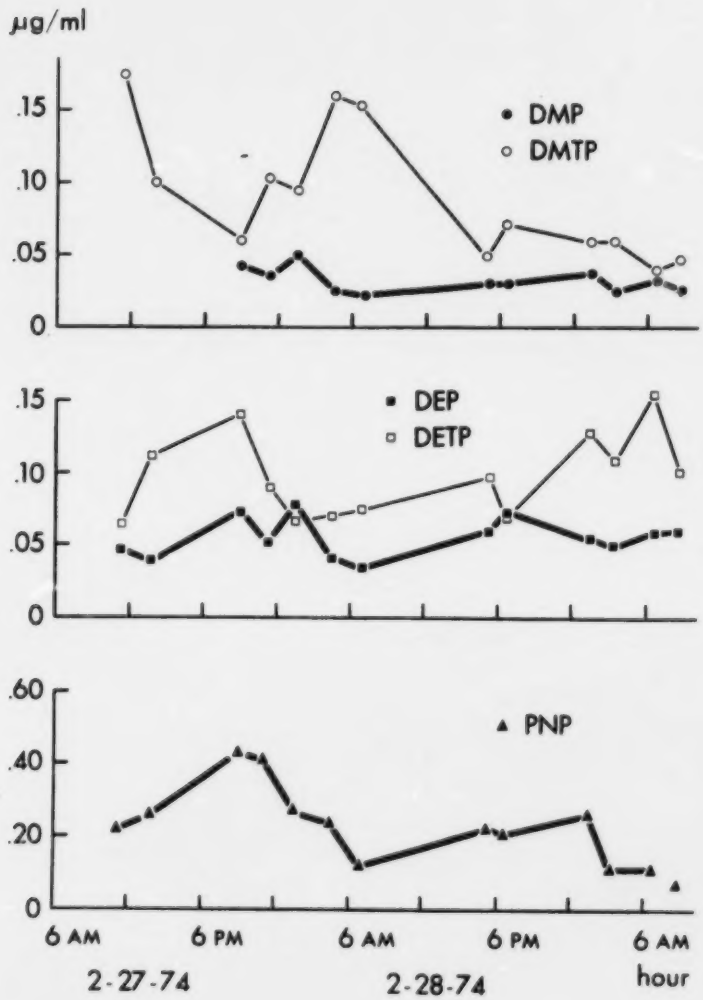


Figure 6. URINARY ALKYL PHOSPHATE AND PARANITROPHENOL EXCRETION IN A HELICOPTER PILOT (FB) DURING AND AFTER APPLICATION OF PARATHION MIXTURES, PHOSDRIN AND CYGON

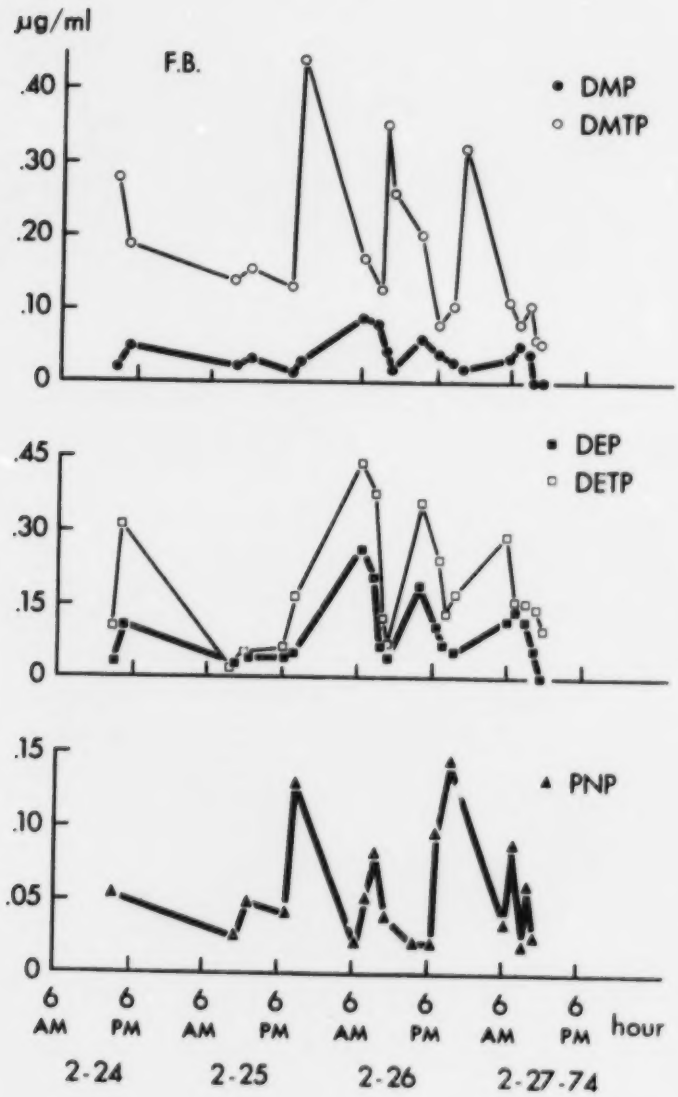


Figure 7. URINARY ALKYL PHOSPHATE AND PARANITROPHENOL EXCRETION IN A HELICOPTER PILOT (GJ) 24 HOURS AFTER OCCUPATIONAL EXPOSURE TO PARATHION, PHOSDRIN AND CYGON

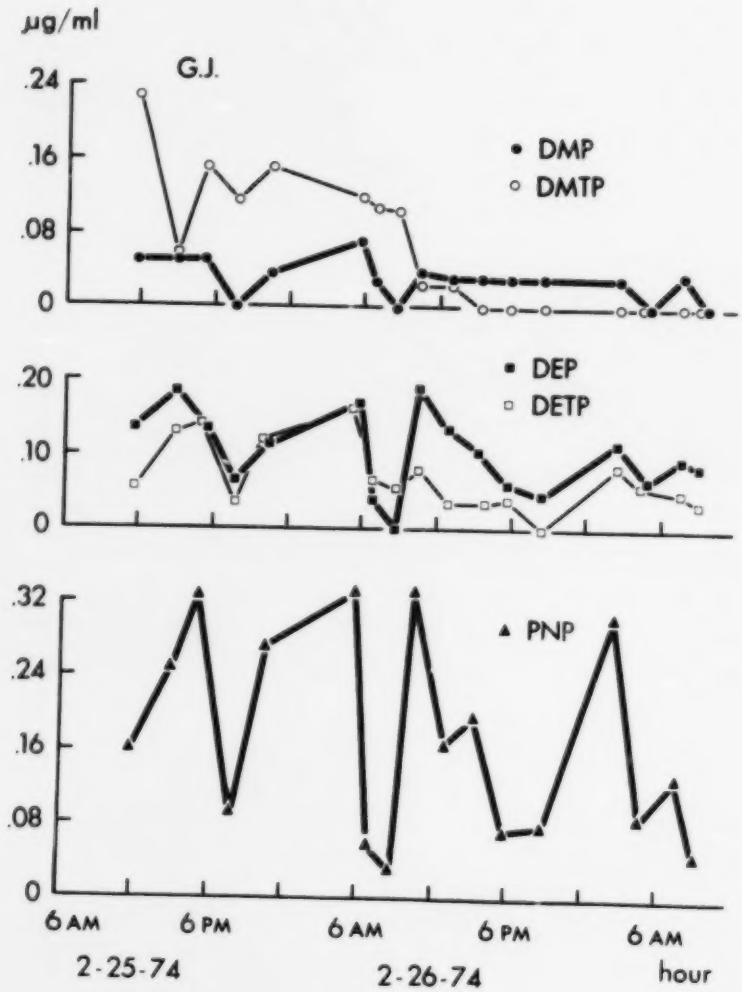


Figure 8. URINARY ALKYL PHOSPHATE AND PARANITROPHENOL EXCRETION RATES IN A HELICOPTER PILOT (DA) 24 HOURS AFTER OCCUPATIONAL EXPOSURE TO PARATHION MIXTURES AND CYGON

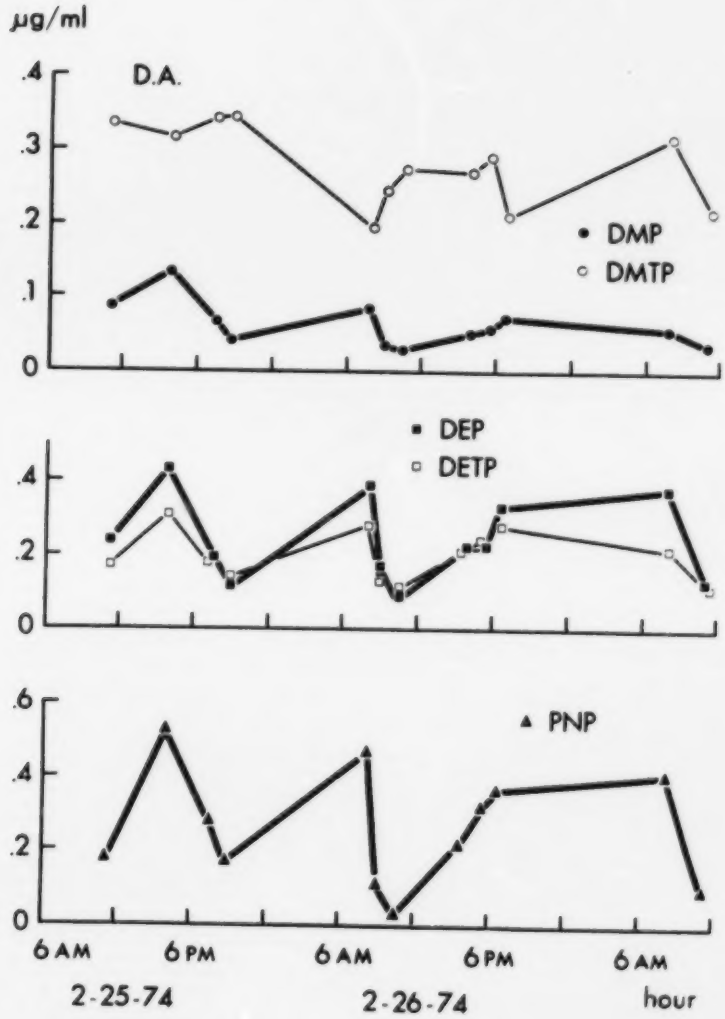


Table 13. LEVELS OF URINARY METABOLITES OF ETHYL PARATHION IN FIRST URINE SAMPLES ANALYZED IN SEVEN CASES OF PARATHION POISONING

Case No.	Name	DEP ug/ml	DETP ug/ml	PNP ug/ml	RBC ChE (ΔpH/hr)	Plasma ChE (ΔpH/hr)
1.	Frank R.	0.46	0.28	0.44	0.21	0.49
2.	Maynard W.	0.44	0.16	0.70	0.37	0.33
3.	Chris W.	7.84	0.09	QNS	0.30##	0.08
4.	Jimmy J.	1.47	0.31	2.26	0.55##	0.20
5.	Matthew P.	2.21	1.48	2.26	0.55##	0.18
6.	Tony B	0.88	0.30	2.14	0.55##	0.55
7.	James D.	0.99	0.10	2.14	0.15	0.10

QNS=Quantity not sufficient
Post-PAM blood

symptomatic cases. Furthermore, D.A. two months later had an RBC ChE of 0.26 ΔpH/hr and plasma ChE of 0.50 ΔpH/hr and it was necessary to remove him from further flying because his RBC ChE had dropped from 0.46 to 0.26 ΔpH/hr. G.J. on the other hand continued to have satisfactory cholinesterase levels when examined two months later (see Table 3).

These qualitative differences of DEP and DETP were compared in the two groups. Table 14 compares the DEP:DETP ratios in 3 poisoned cases and in the occupational group. The mean ratio in the poisoning group was 4.14 (SE=0.70) and the corresponding mean in the occupationally exposed group was 0.88 (SE=0.09). These means are significantly different ($p < .01$) by any of the comparative tests which might be used (t test, Wilcoxson Rank Score, etc.). It has already been mentioned that for the dimethyl alkyl phosphate derivatives compared in these two groups, such a comparison is not truly valid since the comparison in one of different ethyl methyl parathion exposures and unavoidably, the occupationally exposed group had additional exposure to other pesticides. Several of these had DMP or DMTP alkyl phosphate metabolites and would confound the interpretation of the dimethyl data. The qualitative and quantitative differences of the dimethyl alkyl phosphate metabolites is not therefore considered in this study. Paranitrophenol concentrations are almost certainly uniquely representative of ethyl methyl parathion, since there were no exposure to other pesticides which possess this phenolic metabolite in our studies. The comparison of these metabolites were more variable in the two exposure groups and the data suggested that they were less sensitive indicators of parathion exposure, cholinergic symptoms and cholinesterase changes.

Discussion and Conclusion - The urinary metabolite data in these differing exposures to ethyl methyl parathion mixtures suggested that the diethyl alkyl phosphate data were the most important indicators of excessive excessive exposure to this pesticide. As predictors of cholinergic manifestations and severe enzyme inhibition the concentration of DEP was the most important variable. Concentrations of DEP in excess of 0.4 ug/ml were of the order of magnitude which were associated with symptoms and severe degrees of cholinesterase inhibition. The respective ratio of DEP to DETP was another important index of over exposure and ratios of DEP:DETP of 2.0 or greater were usually associated with marked degrees of enzyme inhibition and with poisoning symptoms. Since DEP is reflective of oxon of parathion and DETP is reflective of parathion exposure, then the data suggested that the major contributant to severe cholinesterase inhibition and the occurrence of overt illness in parathion exposure is the amount of paraoxon formed rather than the amount of the original pesticide. This observation is entirely in accord with the known relative toxicity of these two compounds.

Urinary Pesticide Metabolite Studies in Occupational Exposure to Dursban and DDVP

Background Information - Dursban and DDVP are organophosphate insecticides of low mammalian toxicity and to our knowledge there are no reports of human illness and serious cholinesterase inhibition following occupational exposures of these pesticides. Dursban is reported to inhibit plasma ChE only. Urinary pesticide metabolite studies of work exposure to these chemicals cannot be related to illness by cholinesterase changes but serve rather

Table 14. COMPARISON OF $\frac{DEP}{DETP}$ RATIOS WITH URINES FROM PATIENTS WITH PARATHION POISONING WITH ASYMPTOMATIC OCCUPATIONALLY EXPOSED WORKERS. SOUTH FLORIDA. 1974

Name	No. of Urines	DEP:DETP RATIO Mean	Standard Deviation	Standard Error	Minimum	Maximum
A. Poisonings						
TB	6	3.95	3.24	1.32	1.40	9.75
JD	7	7.12	2.58	0.97	4.59	10.33
MP	7	1.79	0.69	0.26	0.90	2.58
Combined	20	4.14+	3.21	0.70	0.90	10.33
B. Exposed Workers						
WG #1	13	0.60	0.24	0.07	0.32	1.16
WG #2	11	0.17	0.21	0.06	0	0.69
FB	19	0.60	0.40	0.09	0	1.73
GJ	16	1.73	1.04	0.08	0	4.42
DA	12	1.17	0.29	0.26	0.64	1.76
Combined	71	0.88+	0.78	0.09	0	4.42

+t test - percent of significance of differences at <0.01

to provide descriptive information of the urinary metabolites with regard to the magnitude and the duration of excretion of these metabolites following known single occupational exposures. Such studies therefore are purely descriptive and this was the goal of the studies undertaken herein. DEP, DETP and 3,5,6-TC pyridinol are the major metabolites of Dursban. DMP is the major alkyl phosphate metabolite of DDVP.

In 1973 a sequential excretion of these two metabolites was studied in two spraymen following a single 4½ hour exposure to 2½% Dursban and 0.75% DDVP. Following this small isolated exposure all three metabolites were still detectable in the urine for 36 hours after exposure. DETP peaked at 6-9 hours after exposures and its concentration was always greater than DEP. 3,5,6-TC pyridinol peaked at 8 hours in one sprayman and 32 hours in another. DEP excretions peaked at 4½ hours after exposure. It was decided to repeat this study in the same sprayman but with a 5½ hour exposure to the same insecticide.

Materials and Methods - After measuring the volume and time of voiding of the first morning's specimen, this was discarded; thereafter, sequential urines were collected from the same two spraymen (M.P. and E.S.) during and after a single 5½ hour exposure to Dursban 2.5% and DDVP 0.75%. Both operators sprayed only on the day of study and had no pesticide exposure on the preceeding days. Both wore respirators and rubber gloves while working. M.P. used a hand sprayer and E.S. used a power sprayer; they sprayed the hold of the ship from 9:00 a.m. to 2:30 p.m. After this they washed and changed their clothing and continued to collect the urine for 32 hours (M.P.) and 29½ hours (E.S.).

Results - Tables 15 and 16 list the concentrations of DMP (DDVP), and DEP, DETP and 3,5,6 TC pyridinol (Dursban) excreted following this single work exposure of these two spraymen. Figures 9 and 10 described the sequential concentration of these four metabolites in ug/ml. Red blood cell and plasma ChE levels for M.P. were 0.76 and 0.76 ΔpH/hr respectively and the same findings for E.S. were 0.69 and 0.76 ΔpH/hr respectively.

Discussion - All four metabolites of the pesticides being applied were identified in both spraymen. DEP concentrations ranged from 0.03 - 0.082 ug/ml for the two men; similarly, DETP levels ranged from 0.06 to 0.18 ug/ml. DETP concentrations were greater than DEP and 3,5,6-TC pyridinol concentrations ranged from 0.014 - 0.380 ug/ml. In contrast, DEP and DETP concentrations did not vary much during these exposures and were of a low order of magnitude. Both the alkyl phosphate and phenolic metabolites, however, were detectable for as long as 30 hours after exposure. 3,5,6-TC pyridinol peaked later than the alkyl phosphate metabolites and was maximal 17 to 27 hours after the end of the exposure in the respective spraymen. The later maximum excretion of this phenolic metabolite was also noted in our studies conducted last year. In contrast, DMP concentrations which ranged from N.D. - 0.3 ug/ml were maximum almost immediately at the end of the work exposure and then declined steadily. Thus, DDVP which had the DMP metabolite appeared to be metabolized at a fast rate than Dursban. This observation reconfirmed the findings of last year.

Urinary Metabolite Studies During and After Early Application of Thimet (Phorate)

Background Information - Phorate is an organophosphate insecticide with an

Table 15. SEQUENTIAL URINARY ALKYL PHOSPHATE AND PHENOLIC EXCRETION RATES IN A STRUCTURAL PEST CONTROL OPERATOR (M.P.) DURING AND AFTER 5 1/2 HOURS APPLICATION OF 2.5% DURSBAN AND 0.75% DDVP

Date 1974	Hour	ml Voided	DMP ug/ ml	DMP ug/vol voided	DMP ug/ hour	DEP ug/ ml	DEP ug/vol voided	DEP ug/ hour	DETP ug/ ml	DETP ug/vol voided	DETP ug/ hour	3,5,6-TC Pyridinol ug/ ml	3,5,6-TC Pyridinol ug/vol voided	3,5,6-TC Pyridinol ug/ hour
11/10	11:50 am	250.0	0.036	9.0	1.90	0.030	7.8	1.60	0.060	15.0	3.10	0.075	18.8	3.90
	2:30 pm	189.0	0.120	22.7	8.50	0.030	5.5	2.10	0.150	28.4	10.60	0.051	9.6	3.60
	5:00 pm	NC	NC	NC	NC	NC	NC	NC	NC	NC	NC	NC	NC	NC
	7:15 pm	150.5	0.117	17.6	7.80	0.030	4.5	2.00	0.050	7.5	3.40	0.045	6.7	3.00
	9:30 pm	200.0	0.030	6.0	2.70	ND	ND	ND	0.090	18.0	8.00	0.023	4.6	2.00
	11:05 pm	218.0	0.030	6.5	4.10	ND	ND	ND	0.100	21.8	13.80	0.019	4.2	2.70
11/11	1:10 am	192.0	0.023	4.4	2.10	ND	ND	ND	0.080	15.4	7.40	0.041	7.8	3.80
	3:00 am	280.0	ND	ND	ND	ND	ND	ND	0.110	30.8	16.80	0.014	3.9	2.10
	5:00 pm	134.0	0.025	3.4	0.24	0.040	5.4	0.38	0.100	13.4	0.96	0.114	15.3	1.10
	10:30 pm	206.0	ND	ND	ND	ND	ND	ND	0.080	16.5	3.00	0.063	13.0	2.40

RBC ChE 0.76 and Plasma ChE 0.76 pH/hr.
NC=Voided at 5:00 pm but urine not collected
ND=Not detectable
Limits of detectability are: DMP 0.03, DEP 0.04, DETP 0.04 and 3,5,6-TC Pyridinol 0.002

Table 16. SEQUENTIAL URINARY ALKYL PHOSPHATE AND PHENOLIC EXCRETION RATES IN A STRUCTURAL PEST CONTROL OPERATOR (ES) DURING AND AFTER 5 1/2 HOURS APPLICATION OF 2.5% DURSBAN AND 0.75% DDVP

Date 1973	Hour	ml Voided	DMP ug/ml	DMP ug/vol voided	DMP ug/hour	DEP ug/ml	DEP ug/vol voided	DEP ug/hour	DETP ug/ml	DETP ug/vol voided	DETP ug/hour	3,5,6-TC Prydinol ug/ml	3,5,6-TC Prydinol ug/vol voided	3,5,6-TC Prydinol ug/hour
11/10	6:21 pm	175	0.300	52.5	UC	0.068	11.9	UC	0.070	12.3	UC	0.090	16.6	UC
	7:45 pm	80	0.258	20.6	14.7	0.082	6.6	4.7	0.170	13.6	9.7	0.129	10.3	7.4
	11:30 pm	200	0.220	44.0	11.7	0.070	14.0	3.7	0.170	34.0	9.1	0.106	21.3	5.7
11/11	8:00 am	175	0.234	41.0	4.8	0.100	17.5	2.1	0.180	31.5	3.7	0.380	66.7	7.8
	2:10 pm	275	0.130	36.0	5.8	ND	ND	ND	0.170	46.8	7.6	0.073	20.0	3.2
	4:50 pm	360	0.056	20.2	7.6	0.078	28.1	10.5	0.160	57.6	21.6	0.185	66.7	25.0
	8:00 pm	220	ND	ND	ND	ND	ND	ND	0.150	33.0	10.4	0.109	24.1	7.6

UC=Unable to calculate previous hour not known
ND=Not detectable
Limits of detectability are: DMP 0.03, DEP 0.04, DETP 0.04 and 3,5,6-TC Pyridinol 0.002

Figure 9. SEQUENTIAL URINARY ALKYL PHOSPHATE AND PHENOLIC EXCRETION RATES IN A STRUCTURAL PEST CONTROL SPRAYER (MP) DURING AND AFTER 5 1/2 HOURS APPLICATION OF 0.75% DDVP AND 2.5% DURSBAN

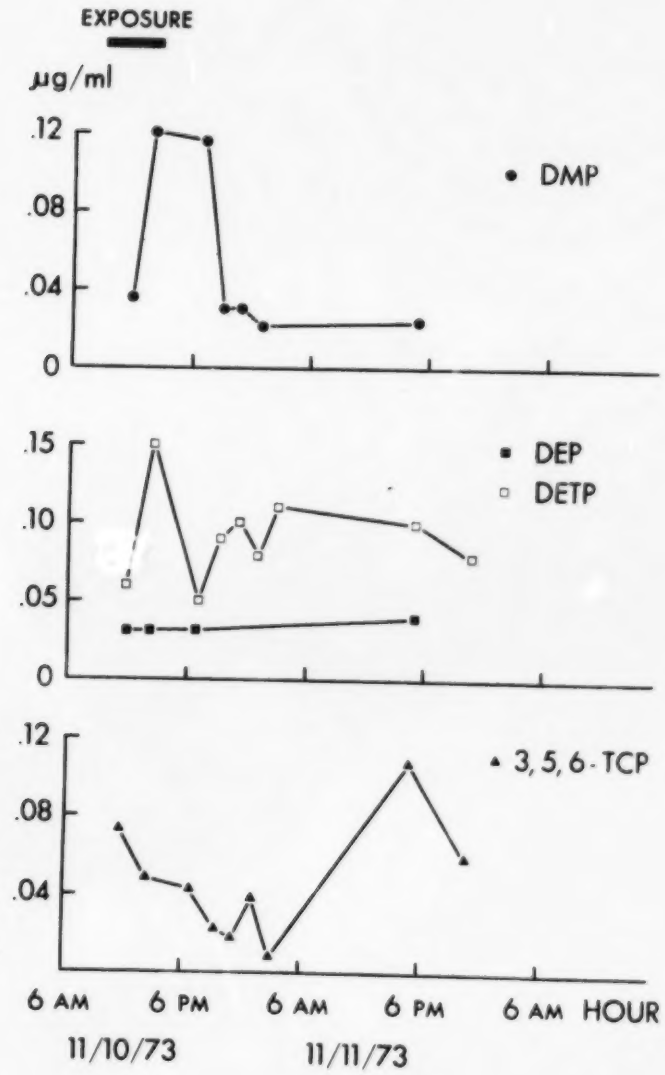
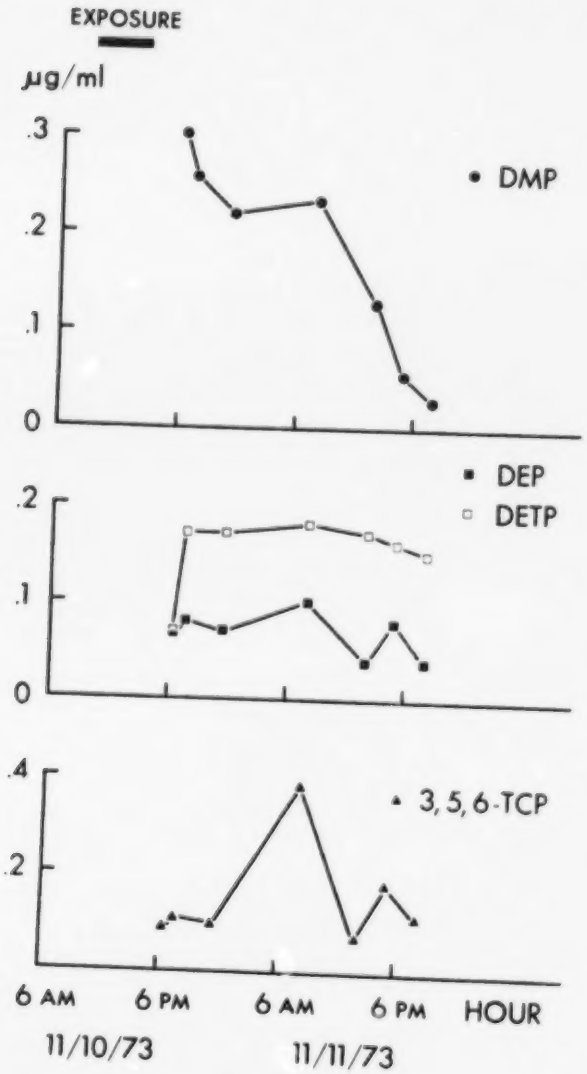


Figure 10. SEQUENTIAL URINARY ALKYL PHOSPHATE AND PHENOLIC EXCRETION RATES IN A STRUCTURAL PEST CONTROL SPRAYER (ES) DURING AND AFTER 5 1/2 HOURS APPLICATION OF 0.75% DDVP AND 2.5% DURSBAN



oral LD50 of 1.1 mg/kg and a dermal LD50 of 2.5 mg/kg; it is therefore a highly toxic pesticide and because of this is infrequently used in agriculture in South Florida except in a granular formulation. An opportunity was afforded us to study the urinary excretion of the alkyl phosphates of Phorate when a 10% granular formulation of this pesticide was aerially applied by a fixed wing aerial applicator during a two hour period. The ordinary alkyl phosphate metabolites of Phorate are DEP, DETP, DEDTP—the latter is present in too low concentrations to be detected under normal occupational exposures, being recognized only in acute poisonings.

Materials and Method - A fixed wing aircraft pilot was visited on 1/19/74 and requested to participate in a pesticide urinary metabolite study. Blood was collected and analyzed that day by the Michel method and showed that his RBC and plasma ChE were 0.60 ΔpH/hr and 0.75 ΔpH/hr respectively. On 1/25/74 the day before the study he was working on the airfield from 8:00 a.m. to 4:00 p.m. but did no flying. On 1/26/75 he discarded his early morning specimen and thereafter voided into a labelled hexane washed jar and noted the amount and time of urine voided. Sequential urines were provided from 7:00 a.m. on 1/26/74 until 10:00 p.m. 1/27/74. On the study day, he worked on the plane at 8:30 a.m. and then between 10:00 a.m. and 12 noon he loaded and applied 10 lb. bags of Thimet (Phorate) granules for a two hour period. The pilot himself opened the bags and then emptied them into the tank of an open cockpit Stearman. He loaded and flew three times and reported that on the third flight he developed a severe headache and felt nauseated. So intense was his headache that it interferred with his flight and so he showered, changed his clothes and went home. He took aspirins, the headache improved and there were no further symptoms. Two days later a second cholinesterase showed that the RBC and plasma cholinesterase were 0.64 and 0.93 ΔpH/hr respectively.

Results - Table 17 shows the urinary concentrations of the alkyl phosphate metabolites from the pilot during and after his application of 10% Thimet. Figure 11 illustrates the sequential excretion of these metabolites in ug/ml.

Discussion - The appropriate metabolites for Phorate that we should study are DEP, DETP, DEDTP, the latter was not detected and this is not uncommon under low level exposure situations. DEP which is reflective of oxon was on all occasions except once less than DETP. This substantiated the negative diagnosis of pesticide related illness. The DETP metabolites very accurately illustrated his work exposure because concentrations of this metabolite started at 1 and rose to 3 after two hours of aerial application of Thimet (Phorate). DEP fluctuations were minimal. The excretion of these metabolites in this setting suggested that there were diurnal variations with high levels found in the morning. It will be noted that traces of DMP and DMTP were identified. We have no explanation for the occurrence of these di-methyl metabolites and can only postulate that these were the result of other organophosphates at the work site and/or traces of contamination on the clothing or in the aircraft.

Urinary Phenolic Studies in Occupational Exposure to Sevin (Carbaryl)

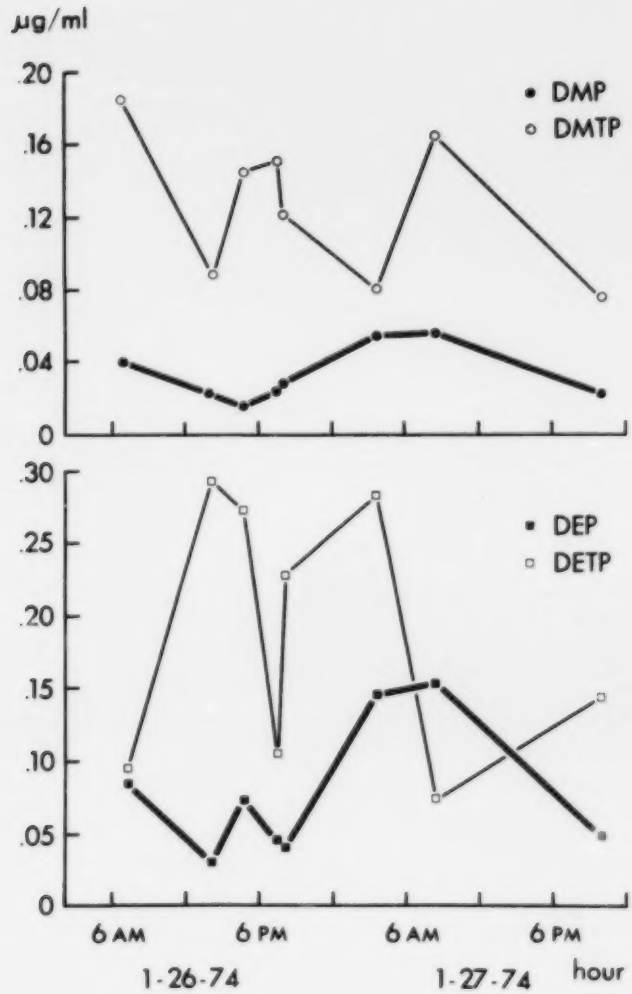
Background Information - Sevin (carbaryl) is not a strong cholinesterase inhibitor and the oral and dermal LD50s of this carbamate insecticide are 850 mg/kg and >4000 mg/kg in male rats respectively. It is thus not a very toxic insecti-

Table 17. URINARY ALKYL PHOSPHATE EXCRETION RATES IN A PILOT (JS) LOADING AND APPLYING PHORATE FOR TWO HOURS

Date 1974	Hour	ml Voided	DMP ug/ml	DMP ug/vol voided	DMP ug/ hour	DMTP ug/ml	DMTP ug/vol voided	DMTP ug/ hour	DEP ug/ml	DEP ug/vol voided	DEP ug/ hour	DETP ug/ml	DETP ug/vol voided	DETP ug/ hour
1/26	7:00 am	300	0.040	12.0	UC	0.176	55.8	UC	0.082	24.6	UC	0.094	28.2	UC
	2:00 pm	350	0.023	8.1	1.2	0.089	31.2	4.4	0.033	11.6	1.7	0.293	102.6	14.7
	4:30 pm	230	0.017	3.9	1.6	0.146	33.6	13.4	0.074	17.0	6.8	0.274	63.0	25.2
	7:30 pm	365	0.023	8.4	UC	0.150	54.8	UC	0.045	16.4	UC	0.106	38.7	UC
	7:45 pm	320	0.029	9.3	UC	0.120	38.4	UC	0.042	13.4	UC	0.226	72.3	UC
1/27	3:30 am	250	0.055	13.8	UC	0.080	20.0	UC	0.146	36.5	UC	0.281	70.3	UC
	8:20 am	150	0.056	8.4	1.7	0.165	24.8	5.1	0.154	23.1	4.8	0.074	11.1	2.3
	?	240	0.018	4.3	UC	0.163	39.1	UC	0.027	6.5	UC	0.043	10.3	UC
	5:15 ?	690	0.015	10.4	UC	0.061	42.1	UC	0.023	15.9	UC	0.145	100.1	UC
	5:20 ?	550	0.014	7.7	UC	0.059	32.5	UC	0.026	14.3	UC	0.150	82.5	UC
	10:00 pm	195	0.022	4.3	UC	0.077	15.0	UC	0.048	9.4	UC	0.141	27.5	UC

UC=Unable to calculate micrograms per hour
?=Cannot establish correct time

Figure 11. URINARY ALKYL PHOSPHATE EXCRETION RATE IN A PILOT (JS) LOADING AND APPLYING PHORATE FOR 2 HOURS



cide and neither illness nor cholinesterase inhibition would be an expected end point in metabolite studies following exposures to 0.5% solution of Sevin. The purpose therefore of this investigation was purely descriptive having special interest in determining the magnitude of alpha naphthol levels and the post exposure peaking and duration of excretion following the completion of work. Alpha naphthol is the phenolic metabolite of carbaryl (Sevin).

Materials and Method - Sequential urinary excretion of alpha naphthol were collected and analyzed from two spraymen working at the Dade County Park Department spraying trees for Dutch Elm disease with a 0.5% solution of Sevin and having a worker exposure period of between 3½ to 6 hours. In addition to spray, exposure occurred at the beginning of the day through the mixing of 30 lbs. of water dispersable powder of 80% Sevin into a 600 gallon capacity tank, and at the end of the day through washing and hosing down the truck. The two spraymen alternated evenly during the spray period between driving the truck or applying the spray. The two men wore short sleeve shirts and wore no gloves and used no respirators. Cholinesterase activities were determined by the Michel method and alpha naphthol was analyzed by the Shafik, Sullivan and Enos method.[9, 10]

Results - The RBC and plasma ChE of R.S. was 1.06 ΔpH/hr and 0.52 ΔpH/hr respectively and for E.O., 0.68 and 1.03 ΔpH/hr respectively. The exposure period and the urinary excretions of alpha naphthol are shown in Table 18. Figure 12 illustrates the daily excretion of alpha naphthol during the study week, and during the weekend period of non-exposure in one sprayman (R.S.). Alpha naphthol was detected in all specimens voided and concentrations ranged from 0.2 - 3.16 ug/ml in the two spraymen. There was a gradual increase of levels from Monday through Thursday with a decline occurring thereafter. Alpha naphthol was still detected 42 hours after R.S.'s last exposure on Friday, 3/29/74 until Sunday, 3/31/74. Thus, the effects of exposure were not totally eliminated by the weekend rest. This would explain why the first collection on Monday morning revealed small traces of alpha naphthol. The weekend data from R.S. also suggests that the optimum time for collecting a post exposure urine was 18 hours after exposure because alpha naphthol was still being detected during the time he was not at work.

Urinary Pesticide Metabolite Studies in Acute and Chronic Exposure of Dichlofenthion

Background Information - Dichlofenthion or VC-13 is an organophosphate which is being used in South Florida as a nemacide and insecticide for the control of chinch bugs. It is readily available in most horticulture and in drug stores, and has an oral LD50 of 270 mg/kg and a dermal LD50 of 6,000 mg/kg in rabbits. Animal studies have shown this pesticide to be soluble in fat. We have identified VC-13 in human fat specimens in a series of five cases of human intoxication, all of which were the result of suicidal ingestion.[12] Such poisonings were very atypical from a clinical point of view, exhibiting a protracted asymptomatic period following ingestion. Initial manifestations were deceiptively mild but were followed in a period of 40-48 hours by evolution of serious and life threatening cholinergic crises. Cholinergic manifestations of waxing and waning in intensity lasted 5-48 days and required continuous atropine therapy and oximes for the entire period. During the middle of this year, we were called in to advise on such a poisoning case because the patient was still exhibiting cholinesterase manifestations on the 12th day after ingestion of VC-13; her red blood cell and plasma cholin-

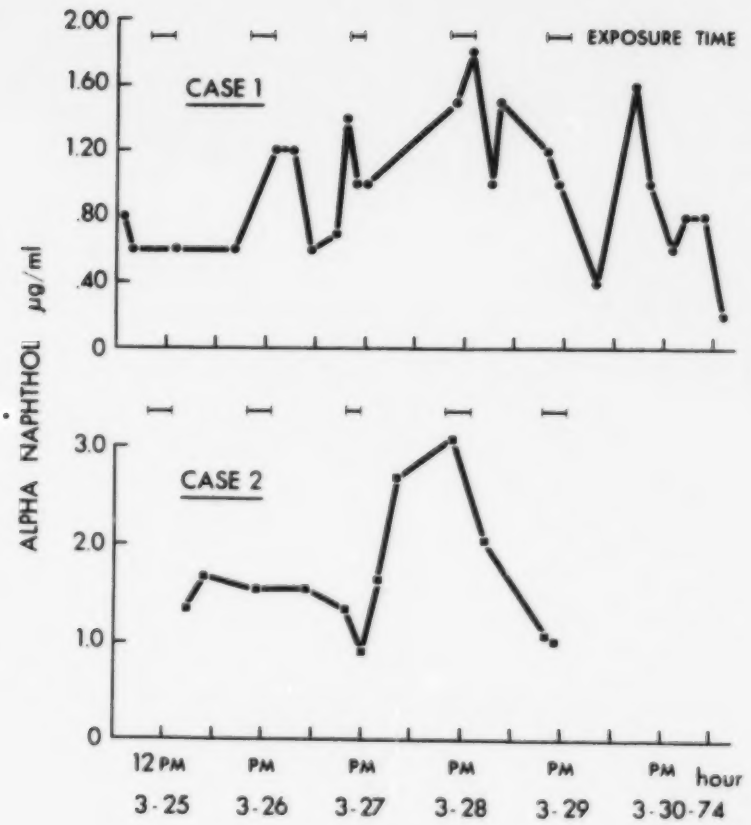
Table 18. ALPHA NAPHTHOL CONCENTRATIONS IN TWO DADE COUNTY PARKS DEPARTMENT SPRAYMEN (R.S. AND E.O) WHO HAD BEEN USING 0.5% SEVIN (CARBARYL)

Date 1974	Exposure history	Robert S.## Hour collected	ml. voided	Alpha Naphthol ug/ml	ug/vol. voided	ug/ hour	Estanislau O.## Hour collected	ml. voided	Alpha Naphthol ug/ml	ug/vol voided	ug/ hour
3/25	8:00 am - 2:00 pm	1:00 am	336	0.80	268.8	UC	5:15 pm	168	1.35	227.14	56.8
		4:32 am	332	0.60	199.2	56.38	9:00 pm	160	1.60	255.36	68.1
		2:50 pm	364	0.60	218.4	UC					
3/26	8:00 am - 2:00 pm	10:45 am	221	0.60	132.6	UC	10:45 am	196	1.55	303.41	52.8
		3:00 pm	165	1.20	198.0	46.59	10:45 pm	142	3.87	549.54	274.8
		6:20 pm	344	1.20	412.8	123.84					
		11.30 pm	350	0.60	210.0	40.65					
3/27	8:00 am - 11:30 am	5:00 am	380	0.70	266.0	48.36	10:00 am	126	1.35	169.60	44.2
		7:00 am	95	1.40	133.0	66.50	12 noon	60	0.86	51.24	25.6
		10:05 am	180	1.00	180.0	58.40	3:25 pm	130	1.70	221.0	64.7
		12 noon	128	1.00	128	66.78	8:30 pm	129	2.72	350.75	69.0
3/28	8:00 am - 2:00 pm	10:15 am	283	1.50	424.5	UC	10:45 am	108	3.16	341.28	68.3
		2:00 pm	114	1.80	205.2	54.72	5:25 pm	150	2.05	307.50	90.0
		6:00 pm	165	1.00	165.0	41.25					
		9:00 pm	110	1.50	165.0	55.0					
3/29	8:00 am - 1:00 pm	8:00 am	140	1.20	168.0	UC	8:00 am	156	1.15	179.40	59.8
		10:15 am	167	1.00	167.0	74.2	10:15 am	134	1.00	134.0	59.6
		7:30 pm	295	0.40	118.0	12.8					
3/30	None	5:00 am	232	1.60	371.20	UC					
		9:00 am	205	1.0	205.0	51.25					
		2:00 pm	375	0.60	225.0	45.0					
		5:00 pm	324	0.80	259.2	86.40					
		10:00 pm	285	0.80	228.0	45.60					
3/31	None	7:00 am	306	0.20	61.2	6.80					

UC=Unable to calculate previous hour not known

RBC ChE 1.06 ΔpH/hr, Pl. ChE 0.52 ΔpH/hr (R.S.) and E.O. RBC ChE 0.68 ΔpH/hr and Pl. ChE 1.03 ΔpH/hr

Figure 12. DAILY EXCRETION OF ALPHA NAPHTHOL (ug/ml) IN TWO SPRAYMEN APPLYING 0.5% SEVIN (CARBARYL)



esterase were almost 100% inhibited. Pesticide residues and urinary metabolite studies were collected in this patient and our involvement in this case provided an excellent opportunity to study the delayed excretions of the urinary metabolite of this less polar organophosphate. The data that follows describes the pesticide residue studies in this extraordinary case, and also presents urinary phenolic metabolite data of a volunteer sprayman (J.D.) who applied VC-13 to his 1/2 acre lawn. The persistence of this organophosphate in air is discussed later in the section under "Air Monitoring". The urinary metabolites for dichlofenthion (VC-13) are DETP and DEP, and 2,4-Dichlorophenol (2,4-DCP).

Materials and Methods - Sequential urines and blood samples were collected from the pesticide poisoning case during the period of her recovery. The intact pesticide was measured in the blood and also in one fat biopsy collected on the 39th day. RBC and plasma ChE determinations were also performed during and after hospitalization and urine was analyzed for the urinary alkyl phosphates by the Shafik et al. method and 2,4-DCP by the Shafik et al method.[4,10] In the field studies, 26 ounces of a 75% solution of VC-13 was applied to a 1/2 acre lawn over 100 minute time period pre, during and post exposure urines were analyzed. Gas chromatographic problems prevented simultaneous analysis of alkyl phosphates. No protective clothing was used and the volunteer sprayed his lawn in a short sleeve shirt, long pants and canvas shoes. Immediately after spraying he showered and changed his clothes.

Results - Tables 19 and 20, and Figures 13 and 14 present the cholinesterase pesticide residue data, phenolic excretion of 2,4-DCP and the urinary alkyl phosphate levels after oral ingestion of VC-13 and during the recovery period. This pesticide was identified in a fat biopsy in a concentration of 34 ppm and the intact pesticide was detectable in the serum for 75 days after exposure. The concentrations of the phenolic metabolite (2,4-DCP) ranged from 35.97 - 0.019 ug/ml. Concentrations of DEP ranged from 5.25 - N.D. ug/ml and for DETP from 15.3 - <0.04 ug/ml. DMP and DMTP traces which probably reflected contamination of dichlofenthion ranged from 0.258 - N.D. ug/ml and from 0.38 - <0.04 ug/ml respectively.

Discussion - The chronicity of the intoxication in this case was its most striking feature. The patient required 2 hourly infusions of atropine IM for 40 days to adequately control ChE symptoms and the alkyl phosphate and phenolic metabolites were detected for a period of up to 75 days and when reexamined on the 129th day levels of all five urinary metabolites had fallen below the limits of detectability.

Table 21 presents the urinary excretion of 2,4-DCP during and after application of VC-13 to a 1/2 acre lawn.

Discussion - Dichlofenthion or VC-13 is one of a group of pesticides being used with increasing frequency in many parts of the world. Some of these are halogenated and include such compounds as Ronnel and Leptophos (Phosvel). In studies elsewhere the partition coefficient for the four organophosphate pesticides were studied: dichlofenthion, Ronnel, Leptophos and parathion. Dichlofenthion had a much higher partition coefficient (1.372×10^5) than the trichloro substitute compound Ronnel (7.5×10^4) and the partition coefficient of dichlofenthion was 20 times greater than parathion. Leptophos was even more fat soluble than dichlofenthion having a partition coefficient of

Table 19. CHOLINESTERASE, PESTICIDE RESIDUES AND PHENOLIC DATA FOLLOWING ORAL INGESTION OF DICHLOFENTHION (VC-13). DADE COUNTY 1974

Date	Hour	RBC ChE ≠	Plasma ChE ≠	VC-13 Pl. (ppb)	VC-13 Fat (ppm)	Hour	ml Voided	2,4-Dichlorophenol ug/ml	2,4-Dichlorophenol ug/vol voided	2,4-Dichlorophenol ug/hour
3/2/74†										
3/14/74	5:45 pm	0.08	0.07	173		5:45 pm	310	7.71	2,390.1	UC
						6:55 pm	90	7.02	631.8	541.5
3/15/74						7:45 pm	180	3.27	588.6	588.6
3/18/74						8:30 am	495	35.97	17,805.2	1,396.5
3/28/74		0.08	0.07	141		2:50 pm	450	6.1	2,745.0	986.8
3/29/74						1:10 ?	?	5.9	?	?
3/30/74						3:20	650	1.16	754.0	UC
3/31/74		0.08	0.07	86	36					
4/2/74						4:10 am	500	3.9	1,950.0	531.8
						9:20 pm	360	5.42	1,951.2	477.9
4/10/74		0.08	0.07	68						
4/11/74						2:45 pm	300	2.82	846.0	253.8
						10:25 pm	290	2.92	846.8	267.4
4/12/74						2:45 pm	360	1.72	619.2	256.2
						10:40 pm	290	2.88	835.2	238.6
4/25/74				11.5						
5/6/74		0.25	0.25	7.6		8:55 am	170	1.04	176.8	96.4
						1:05 pm	197	1.29	253.3	121.6
5/16/74		0.40	0.45	2.2		3:30 pm	186	0.37	69.2	46.1
5/30/74		0.40	0.50	ND		4:20 pm	380	0.19	73.7	73.7
7/9/74		0.65	0.88							

≠Michel method (ΔpH/hr)
† Hospital Admission
UC=Unable to calculate previous hour not known
ND=Not detectable
Limit of detectability for 2,4-D is .021

Figure 13. SERUM CONCENTRATIONS OF DICHLOFENTHION (ppb) AND RED CELL AND PLASMA CHOLINESTERASE LEVELS (ΔpH/hr) DURING RECOVERY FOLLOWING ORAL INGESTION OF DICHLOFENTHION (VC-13)

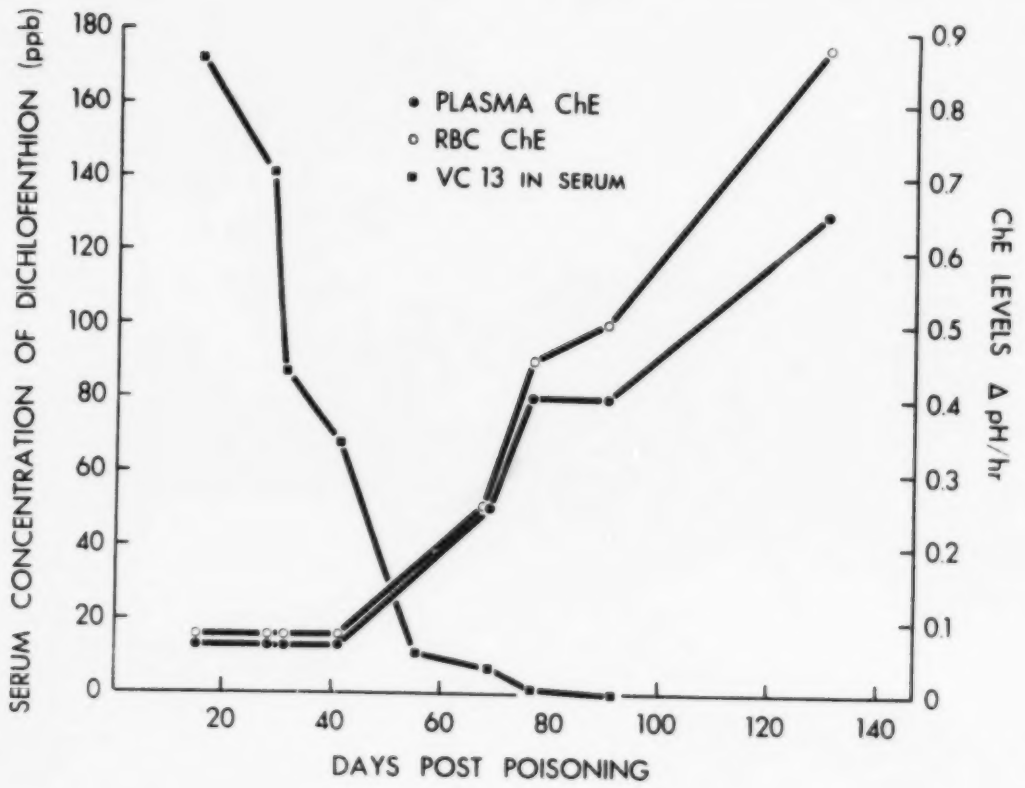


Table 20. URINARY ALKYL PHOSPHATE DURING RECOVERY FOLLOWING ORAL INGESTION OF DICHLOFENTHION. 1974.

Date	Hour	ml Voided	DMP			DMTP			DEP			DETP		
			ug/ml	ug/vv	ug/hr	ug/ml	ug/vv	ug/hr	ug/ml	ug/vv	ug/hr	ug/hr	ug/vv	ug/hr
3/14	5:45 pm	310	0.044	13.6	UC	0.134	41.5	UC	1.53	473.3	UC	4.99	1,545.7	UC
	6:55 pm	90	0.088	7.9	6.8	0.151	13.6	11.7	2.60	233.8	200.4	8.48	762.8	653.8
	7:45 pm	180	0.061	10.98	10.98	0.152	27.4	27.4	1.22	219.2	219.2	1.88	338.4	338.4
3/15	8:30 am	495	0.258	127.7	10.02	0.212	104.9	8.2	5.24	2,593.8	203.4	15.1	7,456.7	584.8
3/18	2:50 pm	450	0.076	34.2	12.1	0.300	135.0	47.7	2.28	1,026.0	362.1	6.58	2,960.1	1,044.7
3/29	1:10 ?	?	0.023	?	?	0.104	?	?	0.80	?	?	3.58	?	?
	3:20 ?	650	0.021	13.7	UC	0.078	50.7	UC	1.12	728.0	UC	0.87	564.2	UC
4/2	4:10 am	500	0.068	34.0	9.3	0.264	132.0	36.0	1.41	705.0	192.3	2.97	1,485.0	405.0
	9:20 pm	360	0.053	19.1	4.7	0.276	99.4	24.3	1.76	633.6	155.2	2.24	807.8	197.8
4/11	2:45 pm	300	0.057	17.1	5.1	0.278	83.4	25.0	1.44	432.0	129.6	1.34	403.2	130.0
	10:25 pm	290	0.060	17.4	5.5	0.254	73.7	23.3	1.57	455.3	143.8	1.25	363.7	114.8
4/12	2:45 pm	360	0.044	15.8	6.6	0.229	82.4	34.0	1.10	396.0	163.9	0.71	255.6	105.7
	10:40 pm	290	0.072	20.9	6.0	0.382	110.8	31.7	1.44	417.6	119.3	0.90	261.0	74.6
5/6	8:55 am	315	0.103	17.5	9.6	0.217	36.9	20.1	0.76	128.5	70.1	0.20	33.3	18.2
	1:05 pm	197	0.072	28.4	13.6	0.188	37.0	17.8	1.00	197.4	94.8	0.21	41.2	19.7
	4:20 pm	380	0.038	13.4	8.9	0.324	60.3	40.2	0.37	68.8	45.9	0.10	17.7	11.8
7/9	12:02 pm	314	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND

UC=Previous hour not known; unable to calculate
?=Hour and amount of voiding not available
ND=Not detectable
Limits of detectability are: DEP 0.02, DMP 0.02, DMTP 0.04 and DETP 0.04

Figure 14. URINARY EXCRETIONS OF ALKYL PHOSPHATES AND PHENOLIC METABOLITES OF DICHLOFENTHION (VC-13) POISONING. DADE COUNTY 1974

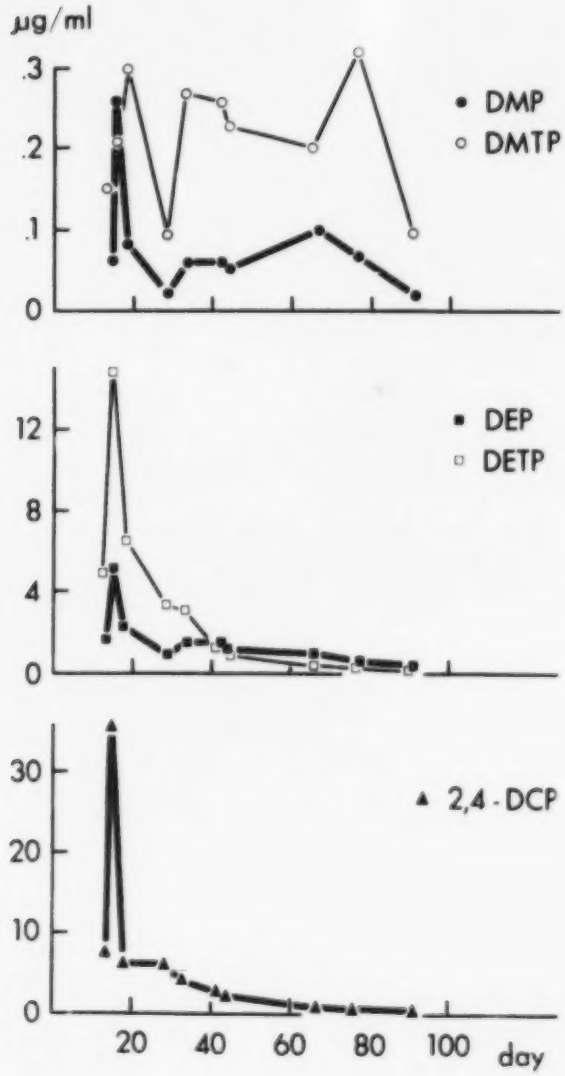


Table 21. 2,4-DICHLOROPHENOL CONCENTRATIONS IN A VOLUNTEER SPRAYMAN (J.D.) USING VC-13 FOR A PERIOD OF 1 HOUR 40 MINUTES

Date	Previous hour	Hour of collection	ml. Voided	2,4-Dichlorophenol ug/ml	ug/vol voided	ug/hour
7/15/74	7:05 am	8:45 am	98	ND*	ND	ND
		10:53 am	236	ND	ND	ND
		3:14 pm	292	0.054	15.77	3.6
		11:40 pm	282	ND	ND	ND
7/16/74		6:50 am	309	ND	ND	ND
		10:43 am	221	ND	ND	ND
		2:00 pm	286	ND	ND	ND
		6:27 pm	164	ND	ND	ND
7/17/74	11:26 pm	7:22 am	234	ND	ND	ND
		8:34 am	82	ND	ND	ND
		1:47 pm	325	ND	ND	ND
		11:43 pm	373	ND	ND	ND
7/18/74		7:25 am	398	ND	ND	ND
		10:45 am	410	ND	ND	ND
7/19/74		7:10 am	372	ND	ND	ND
		11:25 am	422	ND	ND	ND
7/20/74	10:05 am	5:20 pm	440	ND	ND	ND

ND=Not detectable
Limit of detectability 0.02

(2.02×10^6). Based on the partition coefficient data it is our hypothesis that VC-13 is readily absorbed by the fat (possible storage) and through slow mobilization the compound is being released over a period of time, back to the circulatory system where the metabolism changes it to the more toxic oxon and it is this mechanism which we believe accounts for the prolonged symptomatology after exposure.

As is discussed later in the section on "air monitoring", dichlofenthion has been identified in all air samples in the urban environment of Dade County. In view of the fat solubility of this material, if present use patterns continue, it is highly probable that trace amounts of this organophosphate might be detectable in the human adipose pesticide residue profile, especially if lower limits of detectability can be developed.

Pesticide Urinary Metabolite Studies in the General Population

Background Information - Little is known of the magnitude of incidental exposure of the general population of the United States to the non-persistent pesticides. In a presentation at the 167th meeting of the American Chemical Society in Los Angeles, California in 1974, Shafik and Bradway reviewed the very limited data that was available on this topic. Their report included results of earlier studies by Shafik of 14 urines from non-exposed individuals. DETP was not identified and DEP concentrations ranged from 0.04-0.12 ppm. At the low levels of detectability, Shafik and his colleagues were able to detect DEP which was identified in all samples tested. DMP and DMTP were also found in all 14 samples and the range of DMP levels were 0.005-0.04 ppm and this project in 1973 from three non-exposed members of the general population in which none of the alkyl phosphates were identified. He presented limited information from Mrs. Sarah Borthwick of Colorado State University, Fort Collins, Colorado, in her studies from 47 urines from non-exposed Colorado males where the mean levels for DMP and DETP were 0.01 ppm and 0.01 for DEP and DMTP. The percentage frequency of occurrence of both alkyl phosphate and phenolic metabolites from data collected from Ann Yobs of the State Services Program Office of Pesticides, EPA in the human monitoring survey was also discussed. It appeared that from 267 samples 90.3% were positive for DMTP, 70.8% were positive for DETP, 76.7% were positive for DMP and 94% were positive for DEP. In the phenol studies, 96.3% were positive for pentachlorophenol (PCP), 10.9% were positive for alpha naphthol and 6.7% were positive for PNP. The reviewers did not state the lower limits of detectability for the laboratories involved in these studies. In this year's study we planned to conduct additional studies in the general population group but were in part held up in the analysis of the alkyl phosphates because of impurities in the reagents used for preparing the derivatives during the latter part of this year. The data presented hereinafter, therefore, provides more information of phenol prevalence in the general population than alkyl phosphate data.

Materials and Methods - Urines were collected for a variety of subjects having no known occupational exposure to pesticides. Volunteers were asked to note the time and volume of the first morning's specimen and then to discard it. The second voiding was collected in toto in labelled hexane washed jars and time of voiding recorded. Urinary alkyl phosphates were analyzed by the Shafik et al method and the phenols by the Shafik et al method.[10, 8]

Results - The results were expressed in micrograms per milliliter, micrograms per volume voided and micrograms per hour. The lower limits of detectability for DMP and DEP in our laboratory were 0.03 ppm and 0.04 ppm for DETP and DMTP. For the phenols, it was 0.028 ppm for 2,4-DCP, 0.002 ppm for 3,5,6-TC prydinol, 0.001 for 2,4,5-TCP, 0.001 for PCP, 0.009 for PNP, 0.021 for 2,4-D, 0.003 for Silvex, 0.005 for 2,4,5-T, 0.015 for IPP and 0.006 ppm for alpha naphthol.

Table 22a presents the urinary concentrations of the phenols of 38 members of the general population of Dade County, Florida, in 1974. Table 22b presents the urinary concentrations of the alkyl phosphates from seven members of the general population. Table 23 presents the frequency of the urinary metabolites of the general population of Dade County, Florida in 1974.

Discussion - Although this was a small sample and by no means representative of the total general population of Dade County and although concentrations of all of the urinary metabolites identified were uniformly very low, several pesticide metabolites were found in high frequencies. Thus, PCP was found in all 38 samples analyzed and DMTP in all 7 urines tested. PCP appeared to be as ubiquitous as was DDT yet this widespread contamination has received none of the notoriety that DDT has. 3,5,6-TC Pyridinol was the second most commonly found, this urinary phenol being identified in 11 of 38 samples or 29%. This phenol is a urinary metabolite of Dursban which is one of the insecticides commonly used in the homes in Dade County for the control of roaches.

Table 22a. URINARY PESTICIDE CONCENTRATIONS AND RATE OF EXCRETION IN THE GENERAL POPULATION OF DADE COUNTY 1974

Name	Age, Race & Sex	Hr. (min)	ml Void.	Shafik Sullivan Method												Chloroacetic Anhydride Method					
				2,4-DCP	3,5,6-TC Pyrdinol ug/ml	3,5,6-TC Pyrdinol ug/vol voided	3,5,6-TC Pyrdinol ug/hour	2,4,5-T	PCP ug/ml	PCP ug/vol voided	PCP ug/hour	2,4,5-TCP ug/ml	2,4,5-TCP ug/vol voided	2,4,5-TCP ug/hour	PNP	2,4-D	Silvex	IPP	alpha Napthol	Occupation
CM	38 W/F	60	56	ND	0.008	0.45	0.45	ND	0.016	0.89	0.89	ND			ND	ND	ND	ND	ND	Chemist
AB	49 W/F	120	118	ND	ND			ND	0.014	1.65	0.83	ND			ND	ND	ND	ND	*	Chemist
JD	53 W/M	207	278	ND	ND			ND	0.008	2.22	0.65	ND			ND	ND	ND	ND	*	Physician
KF	W/F			ND	ND			ND	0.012	1.4	0.27	ND			ND	ND	ND	ND	ND	Lab Tech
OF	W/M			ND	ND			ND	0.018	2.2	0.24	ND			ND	ND	ND	ND	ND	Lab Tech
CR	32 W/F	128	34	ND	0.006	0.240	0.095	ND	0.016	0.54	0.25	ND			ND	ND	ND	ND	ND	Cashier
LR	69 W/F	172	262	ND	ND			ND	0.002	0.32	0.18	ND			ND	ND	ND	ND	ND	Housewife
JT	23 W/M	105	120	ND	ND			ND	0.004	0.48	2.27	ND			ND	ND	ND	ND	ND	Lab Tech
PT	23 W/F	193	160	ND	ND			ND	0.002	0.32	0.09	ND			ND	ND	ND	ND	ND	Secretary
MB	74 W/F	120	144	ND	0.006	0.864	0.432	ND	0.004	0.57	0.38	ND			ND	ND	ND	ND	ND	Housewife
LR	28 W/F	480	60	ND	ND			ND	0.020	1.2	0.15	ND			ND	ND	ND	ND	ND	Housewife
CR	4 W/M	480	110	ND	ND			ND	0.016	1.76	0.22	ND			ND	ND	ND	ND	ND	
AR	35 W/M	480	304	ND	ND			ND	0.022		0.83	ND			ND	ND	ND	ND	ND	Hairdresser
LP	46 W/M	390	108	ND	ND			ND	0.016	1.73	0.26	ND			ND	ND	ND	ND	ND	Draftsman
PL	10 W/M	570	306	ND	ND			ND	0.004	1.22	0.13	ND			ND	ND	ND	ND	ND	Student
AL	42 W/F	450	152	ND	0.008	1.22	0.162	ND	0.006	0.91	0.12	0.10	1.52	0.20	ND	ND	ND	ND	ND	Seamstress
AM	39 B/M	440	158	ND	0.008	1.26	0.17	ND	0.010	1.58	0.22	ND			ND	ND	ND	ND	ND	PhD Director
CN	48 B/F	60	38.5	ND	0.024	0.92	0.92	ND	0.010	0.39	0.39	ND			ND	ND	ND	ND	ND	Counselor
RK	36 W/M	112	101	ND	0.006	0.61	0.32	ND	0.012	1.21	0.65	ND			ND	ND	ND	ND	ND	Manager
FA	29 B/M	85	56.8	ND	0.006	0.34	0.24	ND	0.006	0.34	0.24	ND			ND	ND	ND	ND	ND	Lab Tech
SK	32 W/F	90	56	ND	ND			ND	0.010	0.56	0.37	ND			ND	ND	ND	ND	ND	Secretary
LW	40 W/M	295	434	ND	ND			ND	0.002	0.87	0.18	0.014	6.08	1.24	ND	ND	ND	ND	ND	Lab Superv.
MT	24 B/F	60	61	ND	ND			ND	0.008	0.49	0.49	ND			ND	ND	ND	ND	ND	Lab Tech
JG	21 W/M	240	129	ND	0.032	4.13	1.03	ND	0.006	0.77	0.19	ND			ND	ND	ND	ND	ND	Mail Clk.
CH	55 W/M	360	409	ND	ND			ND	0.004	1.64	0.27	ND			ND	ND	ND	ND	ND	Mail Superv.
FR	51 W/M	110	266	ND	ND			ND	0.006	1.60	0.87	ND			ND	ND	ND	ND	ND	Univ. Prof.
ER	33 B/M	140	328	ND	ND			ND	0.001	0.46	0.19	ND			ND	ND	ND	ND	ND	Lab Tech
MC	26 B/M	240	158	ND	ND			ND	0.012	1.89	0.47	0.004	0.63	0.16	ND	ND	ND	ND	ND	Airline Mech
AC	21 B/F	240	97	ND	ND			ND	0.010	0.97	0.24	ND			ND	ND	ND	ND	ND	Secretary
IW	19 B/F	135	61	ND	ND			ND	0.006	0.36	0.16	ND			ND	ND	ND	ND	ND	Secretary
JP	48 B/M	135	109	ND	ND			ND	0.006	0.65	0.29	ND			ND	ND	ND	ND	ND	Unemployed
JW	16 B/M	80	27	ND	ND			ND	0.020	0.54	0.41	0.004	0.11	0.08	ND	ND	ND	ND	ND	Unemployed
AC	21 B/M	150	56	ND	0.008	0.45	0.18	ND	0.006	2.34	0.34	ND			ND	ND	ND	ND	ND	Lab Tech

Table 22 . (Continued)

Table 22b.

Name	Age, Race Sex	Hr (min)	ml void	2,4 DCP	3,5,6-TCP ug/ml	3,5,6-TCP ug/vv	3,5,6-TCP ug/hr	2,4,5-T	PCP ug/ml	PCP ug/vv	PCP ug/hr	2,4,5-TCP ug/ml	2,4,5-TCP ug/vv	2,4,5-TCP ug/hr	PNP	2,4-D	Silvex	IPP	α Naphthol	Occupation
R.P.	59 B/F	245	211	ND	ND	ND	ND	ND	0.002	0.38	0.09	ND			ND	ND	ND	ND	ND	Reg. Nurse
R.C.	36 B/M	140	211	ND	ND	ND	ND	ND	0.002	0.38	0.16	ND			ND	ND	ND	ND	ND	Reg. Nurse
J.T.	45 B/M	295	66	ND	ND	ND	ND	ND	0.006	0.38	0.08	0.024	1.61	0.33	ND	ND	ND	ND	ND	Plasterer
M.H.	23 B/F	220	106	ND	ND	ND	ND	ND	0.023	2.48	0.68	ND			ND	ND	ND	ND	ND	Pkg. Clerk
A.J.	B/F	300	92	ND	ND	ND	ND	ND	0.002	0.18	0.04	ND			ND	ND	ND	ND	ND	Housewife

B. ALKYL PHOSPHATE DATA

Name	Age, Race Sex	Hr (min)	ml void	DMP ug/ml	DMP ug/vv	DMP ug/hr.	DMTP ug/ml	DMTP ug/vv	DMTP ug/hr.	DEP ug/ml	DEP ug/vv	DEP ug/hr.	DETP ug/ml	DETP ug/vv	DETP ug/hr.	Occupation
C.M.	38 W/F	60	56	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	Chemist
A.B.	49 W/F	120	118	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	Chemist
J.E.D.	53 W/M	190	154	ND	ND	ND	ND	ND	ND	ND	ND	ND	0.073	11.2	3.7	Physician
J.D.	50 W/M	217	278	ND	ND	ND	0.16	15.2	9.1	ND	ND	ND	0.05	4.9	2.9	Investigator
J.A.	72 W/M	?	?	ND	?	?	ND	?	?	ND	?	?	ND	?	?	Retired
M.P.	4½ W/M	60	15	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	Child
A.S.	51 W/F		314	ND	ND	ND	[redacted]	ND	ND	ND	ND	ND	ND	ND	ND	Housewife

*C.M. 0.67 ug/ml, 37.5 ug/vv and 37.5 ug/hr of 2,4-DCP
**A.B. 0.13 ug/ml, 1.5 ug/vv and 0.8 ug/hr and J.D. 0.14 ug/ml and 3.9 ug/vv and 1.13 ug/hr of α naphthol
?=Unable to calculate hour and volume of urine unknown
ND=Not detectable

Limits of detectability are: DMP 0.03, DMTP 0.04, DEP 0.04 and DETP 0.04 for alkyl phosphates
2,4-DCP 0.028; 3,5-6 TC Pyridinol 0.002; 2,4,5-TCP 0.001; PCP 0.001; PNP 0.009;
2,4-D 0.021; Silvex 0.003; 2,4,5-T 0.005; IPP 0.015; and alpha naphthol 0.006
for phenols

Table 23. FREQUENCY OF OCCURRENCE OF PESTICIDE URINARY METABOLITES (ug/ml) IN THE GENERAL POPULATION OF DADE COUNTY, FLORIDA.

Metabolites	No. of persons tested	No. positive	Per cent positive	Ranges
A. PHENOLS				
Pentachlorophenol	38	38	100	0.001-0.02
3,5,6-TC Pyridinol	38	11	29	<0.002-0.03
2,4,5-TCP	38	6	16	0.002-0.02
α napthol	38	2	5	0.13-0.14
2,4-Dichlorophenol	38	1	3	
2,4,5-T	38	0		
Paranitrophenol	38	0		
2,4-D	38	0		
Silvex	38	0		
Isopropoxyphenol	38	0		
B. ALKYL PHOSPHATES				
Dimethyl phosphate	7	3	43	<0.03
Dimethyl thiophosphate	7	7	100	<0.04 - 0.16
Diethyl phosphate	7	4	57	<0.03
Diethyl thiophosphate	7	6	86	<0.04 - 0.3

SECTION V

Background Information - This is a continuation of air monitoring activities conducted in 1973 in the South Florida area. The data last year indicated a changing profile of the ambient air in South Florida. Minimal traces of organophosphates were identified, in contrast to the usual presence of organochlorine pesticides. DDT was not identified in any of the samples although p,p'-DDE was found in trace amounts. The three sample sites last year were a suburban site, Everglades National Park and Miami International Aiport. It was decided to continue these activities in 1974 in eight different sites and in addition to obtain a qualitative assessment of the pesticide profile in the ambient air by experimenting with the use of an ethylene glycol impregnated nylon organdy screen 0.5 M^2. These activities have assumed increased importance since we have been informed that these are one of the few ongoing pesticide monitoring studies of air being conducted at present in the United States.

Description of Phases

Phase I - Continuation of sampling sites as in 1973.
Phase II - Relocation of work sites.
Phase III - Pesticide studies of air concentrations of VC-13.
Phase IV - Nylon cloth screen experimental studies.

Materials and Methods - A double impinger system was used in each sampler and 100 ml of ethylene glycol placed in each impinger. During the first 12 hour period air was drawn through the double impinger system and at the completion of this 12 hour period, a second sample of air was drawn through the second impinger system. The vacumn pump was run at all full capacity during the 24 hour period. The 24 hour volume collection of air was recorded as was the wind direction and velocity. This 24 hour sample was then transferred to the University for analysis. The analytical method used to measure the air samples was the Sherma and Shafik method.[13] A slight modification was made in the second and third fractions by the addition of 5 and 10 drops of keeper, this prevented the loss of the organophosphate compounds. For the testing of the nylon organdy screen the Tesari and Spencer method was used.[14] The cloth screen was washed with methylene chloride and then with hexane. It was then cut into 1/2 square meters and impregnated with a 10% ethylene glycol in acetone. Three screens were treated in this way. The first screen was placed in a frame 6 feet from the ground in a vertical position facing east which was the direction of the wind at the time of the study, then left for 24 hours and placed in a hexane washed jar and transferred to our laboratory for analysis. The cloth screens were analyzed by the Sherma and Shafik method.[13] Analysis of the screen was seriously complicated by the identification of numerous interfering peaks due to background impurities in the material, therefore, a third screen was spiked with 11 pesticides and the percentage recovery of these were as follows:

I Fraction		II Fraction		III Fraction	
Heptachlor	107%	Lindane	83%	Diazinon	103%
Aldrin	90%	Hep. Epox.	90%	Malathion	108%
p,p'-DDE	106%	Dieldrin	96%		
p,p'-DDD	94%	E. Parathion	95%		
p,p'-DDT	95%				

Results - Tables 24 and 25 list the concentrations of pesticides in air from 14 collections from the suburban site in downtown Miami in 1974 and from the Everglades National Park. Table 26 lists the pesticide concentrations from the Bimini control sampling site and from selected pesticide work sites in South Florida where occupational studies of workers were being conducted during this contract year. Highest serial concentrations of pesticides in all of the air samples were obtained inside a formulating plant. Table 27 compares levels from inside a formulating plant and from a storage shed where packaged pesticides are stored.

The mean and ranges of pesticide concentrations in air and frequency of occurrence of all the pesticides tested are in the suburban site and the Everglades are shown in Tables 28 and 29 respectively. The concentrations of pesticides when measured by the cloth screen are shown in Table 30. Comparisons of these residue collections by the MRI double impinger air sampler and the cloth screen are shown in Table 31.

The persistence of VC-13 in air was studied by repeated sampling by air above the lawn site where 26 ounces of a 75% VC-13 solution has been applied. The air concentrations of the pesticide 10, 27 and 50 days after application are shown in Table 32.

Discussion - From a qualitative point of view, traces of a variety of pesticides were found with regular frequency in both downtown Miami and the Everglades. At the former site VC-13, Dursban and Lindane were found in all samples collected. Heptachlor, Diazinon and Dieldrinwere the pesticides next most commonly found in the urban site. Parathion, DDT and its metabolites were never found at either site. Alpha and gamma chlordane and Ronnel were identified for the first time in the ambient air of South Florida. In the Everglades, alpha-BHC, Diazinon, Lindane and Dursban were the pesticides most frequently identified. The detection of Malathion in both areas was seasonal and coincided with the use of this insecticide for mosquito control during the hot and humid months. Pesticides were identified more frequently in the air of the suburban environment of Miami than in the Everglades.

The concentrations of pesticides in air were not high except in the areas close to pesticide formulating plants or areas where pesticides are mixed for aerial application. In the formulating plant, very high concentrations of a wide variety of pesticides were identified, a finding which emphasized the importance of wearing protective clothing and respirators. The air concentrations in the plant were strikingly different than those seen in a pesticide storage shed where the compounds were sealed and where there was no preparation of formulated products. The air profile of the pesticides in the formulating plant is not only illustrative of the chemicals in greatest demand in the area, but the data also illustrate the opportunity for cross contamination of products during the formulating process. In contrast to these urban and industrial sites, the island of Bimini in the Bahamas was almost totally free from aerial contamination by pesticides.

The data from the cloth screen was really qualitative rather than quantitative. If the pesticides trapped by the screen are compared to those identified

Table 24. PESTICIDE CONCENTRATIONS IN AIR SAMPLES (ng/m^3) FROM SUBURBAN SITE IN SOUTH FLORIDA. 1973-74.

Pesticide	S #3	S #4	S #7	S #11	S #13	S #17	S #19	S #21	S #24	S #27	S #29	S #32	S #36	S #38
VC-13	0.6	0.8	0.8	0.6	0.6	1.0	1.0	0.5	65.0	5.0	1.3	0.7	0.7	0.4
Dursban	1.8	4.4	3.7	3.3	1.4	2.5	2.4	2.3	2.0	2.8	1.5	1.4	1.9	2.2
Diazinon	ND	1.2	1.7	1.1	3.3	2.3	1.0	1.2	2.2	0.5	1.7	1.0	2.1	2.1
Malathion	ND	ND	ND	ND	ND	ND	4.3	28.1	18.2	ND	ND	ND	ND	2.0
α BHC	0.2	1.4	1.8	0.4	0.5	0.5	0.2	0.3	0.5	0.4	0.6	0.5	0.8	0.6
Heptachlor	ND	1.8	2.2	1.2	1.8	0.9	0.7	1.0	1.5	0.9	1.5	1.2	0.6	1.4
Aldrin	ND	ND	ND	ND	1.1	ND	ND	1.1	ND	ND	ND	ND	ND	ND
Lindane	0.2	2.5	1.7	1.5	0.4	0.3	0.2	0.3	0.3	0.3	0.3	0.5	0.3	0.3
Dieldrin	ND	0.5	ND	0.5	0.4	0.5	0.3	0.9	0.6	0.3	0.7	1.0	0.3	0.6
Ronnel	ND	ND	ND	0.6	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
α Chlordane								ND	ND	ND	ND	ND	ND	0.9
γ Chlordane								ND	ND	ND	ND	ND	ND	1.4
m^3 Total Air Volume	51	51	49	54	51	49	53	51	51	52	50	49	54	50
Date Collected (24 hrs)	12/2/73	2/7/74	2/26	3/22	4/30	5/28	6/25	7/8	7/26	8/7	9/3	9/17	10/14	11/5
Wind-Start	NE-N	E-SE	N-NW	E-SE	SE	S-SW	S-SW	E-NE	E-SE	Calm	S	E	SE	E-SE
Finish	SE	E-SE	N-NW	SW	SE-E	Calm	S	E	E-SE	E-SE	SE	E	SE	

S=Sample
ND=Not detectable

All the following pesticides were not detected (ND) in the 14 samples: p,p'-DDE, p,p'-DDT, o,p'-DDE, β-BHC, HCB, Endrin, Trithion, Ethion, Methyl Parathion, Ethyl Parathion, Phorate, Methyl Bromophos, Fenthion, Chlorobenside, Methyoxychlor and Toxaphene.

Table 25. PESTICIDE CONCENTRATIONS IN AIR SAMPLES (ng/m^3) FROM EVERGLADES SITE IN SOUTH FLORIDA. 1973-74

Pesticide	S #1	S #2	S #5	S #8	S #10	S #14	S #16	S #18	S #20	S #26	S #30	S #35	S #39	S #41
Dursban	2.0	2.6	ND	ND	1.1	0.5	0.5	0.3	0.3	ND	1.0	1.1	ND	ND
Diazinon	1.1	ND	ND	1.3	0.8	ND	0.5	0.2	0.2	ND	0.3	1.9	1.1	1.5
Malathion	ND	ND	ND	ND	ND	ND	ND	ND	38.1	ND	ND	ND	ND	ND
α BHC	0.4	0.5	0.2	0.2	0.1	0.4	0.3	0.2	0.3	0.3	0.9	0.6	0.4	0.5
Heptachlor	ND	ND	ND	ND	ND	ND	ND	ND	ND	0.1	1.7	0.3	0.4	0.2
Aldrin	ND	ND	ND	ND	ND	0.5	ND	ND	ND	ND	ND	ND	ND	ND
Lindane	0.2	0.3	0.1	0.2	0.2	0.5	ND	ND	0.2	0.1	0.3	ND	0.1	ND
Dieldrin	ND	ND	ND	ND	0.3	0.2	ND	ND	ND	ND	0.5	ND	0.3	ND
Ronnel	ND	ND	ND	ND	ND	ND	ND	ND	0.9	ND	ND	ND	ND	ND
α Chlordane	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	0.3	ND
γ Chlordane	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	0.5	ND
m^3 Total Air Volume	36	28	45	43	47	43	43	41	41	41	38	37	38	38
Date Collected	11/4/73	12/2	2/7/74	2/26	3/22	4/30	5/28	6/25	7/8	8/7	9/3	10/7	11/5	12/9
Wind - Start	N-NE	N-NE	E-SE	N-NW	E	E-SE	S-SE	S-SE	N-NE	SE-E	SE	W-NW	E	N-NW
Finish	SW	NE	E-SE	N	W	NW	S-SW	S-SW	SE	E	SE	N	SE	N-NE

S=Sample
ND=Not detectable

All the following pesticides were not detected (ND) in the 14 samples: VC-13, p,p'-DDE, p,p'-DDT, o,p'-DDE, β-BHC, HCB, Endrin, Trithion, Ethion, Ethyl Parathion, Methyl Parathion, Phorate, Methyl Bromophos, Fenthion, Chlorobenside, Phosdrin, Methoxychlor, and Toxaphene

Table 26. PESTICIDE CONCENTRATION IN AIR SAMPLES (ng/m^3) FROM BIMINI CONTROL SAMPLING SITE AND FROM SELECTED PESTICIDE WORK SITES IN SOUTH FLORIDA. 1974

Pesticides	Bimini				Allied Hel.		Tri-State		Miami Airport		Shed
	S #6	S #9	S #12	S #15	S #22	S #33	S #23	S #31	S #25	S #37	S #40
VC-13	ND	ND	ND	ND	ND	ND	0.6	ND	0.6	ND	ND
Dursban	ND	ND	0.8	0.5	0.5	1.6	ND	0.8	2.0	12.9	ND
Diazinon	ND	ND	0.4	ND	48.7	37.6	0.5	0.8	0.9	1.0	27.3
Malathion	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	1.6
α BHC	0.1	ND	0.1	ND	1.1	0.8	1.1	1.0	0.3	0.8	2.5
Heptachlor	ND	ND	ND	ND	ND	I	0.4	1.5	0.8	0.3	I
Lindane	ND	ND	ND	ND	0.4	0.3	19.8	1.2	0.2	0.7	1.1
Dieldrin	ND	ND	0.2	ND	ND	ND	1.6	2.2	ND	0.4	33.2
Endrin	ND	ND	ND	ND	ND	ND	2.3	1.0	ND	ND	5.6
Ethion	ND	ND	ND	ND	ND	ND	ND	17.5	ND	ND	ND
Ethyl Parathion	ND	ND	ND	ND	135.7	196.0	12.1	5.9	ND	ND	48.9
Methyl Parathion	ND	ND	ND	ND	59.1	21.0	4.1	ND	ND	ND	4.9
Phorate	ND	ND	ND	ND	0.8	0.3	ND	0.3	ND	ND	0.2
Phosdrin	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	1.3
α Chlordane	ND	ND	ND	ND	ND	ND	ND	7.6	ND	ND	ND
γ Chlordane	ND	ND	ND	ND	ND	ND	ND	6.6	ND	ND	ND
Toxaphene	ND	ND	ND	ND	ND	P	ND	ND	ND	ND	P
Total air collected m^3	51	53	49	53	39	40	36	39	43	52	52
Date Collected	2/11	Mar.	3/22	5/24	7/12	9/23	7/26	9/16	8/5	10/29	11/13
Wind-Start	N-NE	NA	S	S-SE	Calm	E	Calm	E	SE	E-SE	Calm
Finish	NE	NA	NA	S-SE	E-SE	NE	E-SE	E	E-SE	E-NE	Calm

S=Sample
ND=Not detectable
NA=Not available
I=Interference
P=Present

All the following pesticides were not detected (ND) in the 11 samples: Aldrin, p,p'-DDE, p,p'-DDT, o,p'-DDE, B-BHC, HCB, Trithion, Ronnel, Methyl Bromophos, Fenthion, Chlorobenside, Methoxychlor

Table 27. INDOOR CONCENTRATIONS OF PESTICIDES (ng/m^3) AT TWO SELECTED SITES IN SOUTH FLORIDA, 1974.

Pesticides	Storage Shed South Bay S #40	Formulating Plant Goulds S #28
VC-13	ND	10.3
Dursban	ND	1,417.0
Diazinon	27.3	2,078.0
Malathion	1.6	7,305.0
α BHC	2.5	1,913.0
Heptachlor	I	22,171.0
Aldrin	ND	437.0
Lindane	1.1	2,082.0
Dieldrin	33.2	418.0
Endrin	5.6	107.0
Trithion	ND	3.5
Ethion	ND	4.0
Ronnel	ND	10.2
Ethyl Parathion	48.9	557.0
Methyl Parathion	4.9	30.7
Phorate	0.2	0.6
Methyl Bromophos	ND	1,544.0
Phosdrin	1.3	ND
Methoxychlor	ND	254.0
α Chlordane	ND	1,535.0
γ Chlordane	ND	2,303.0
p,p'-DDT	ND	278.0
Toxaphene	P	ND
Total air collected m^3	52	28
Date collected (24 hrs.)	11/12/74	8/15/74

S=Sample
ND=Not detectable
I=Interference
P=Present

All the following pesticides were not detected (ND) in the two samples: p,p'-DDE, o,p'-DDE, β-BHC, HCB, Fenthion, Chlorobenside

Table 28. MEAN, RANGES AND FREQUENCY OF OCCURRENCE OF PESTICIDES IN 14 AIR SAMPLES (ng/m^3) AT SUBURBAN SITE. 1973-74.

Pesticide	Mean	Range	Frequency of Occurrence
VC-13	0.7	0.4 - 1.0	11/11*
Dursban	2.4	1.4 - 4.4	14/14
Diazinon	1.5	ND - 3.3	13/14
Malathion	3.8	ND - 28.1	4/14
α BHC	0.6	0.2 - 1.8	14/14
Heptachlor	1.2	ND - 2.2	13/14
Aldrin	0.2	ND - 1.1	2/14
Lindane	0.7	0.2 - 2.5	14/14
Dieldrin	0.5	ND - 1.0	12/14
Ronnel	0.04	ND - 0.6	1/14
α Chlordane	0.1	ND - 0.9	1/14
γ Chlordane	0.1	ND - 1.4	1/14

ND=Not detectable
*Three VC-13 samples were excluded because they were collected just after application of this pesticide.

All the following pesticides were not detected (ND) in the 14 samples: p,p'-DDE, p,p'-DDT, o,p'-DDE, β-BHC, HCB, Endrin, Trithion, Ethion, Ethyl Parathion, Methyl Parathion, Phorate, Methyl Bromophos, Fenthion, Chlorobenside, Methoxychlor, and Toxaphene.

Table 29. MEAN, RANGES AND FREQUENCY OF OCCURRENCE OF PESTICIDES IN 14 AIR SAMPLES (ng/m^3) AT THE EVERGLADES SITE. 1973-74

Pesticides	Mean	Range	Frequency of Occurrence
Dursban	0.67	ND - 2.6	9/14
Diazinon	0.64	ND - 1.9	10/14
Malathion	2.22	ND - 38.1	1/14
α BHC	0.38	0.1 - 0.9	14/14
Heptachlor	0.19	ND - 1.7	5/14
Aldrin	0.04	ND - 0.5	1/14
Lindane	0.16	ND - 0.5	10/14
Dieldrin	0.09	ND - 0.5	4/14
Ronnel	0.64	ND - 0.9	1/14
α Chlordane	0.02	ND - 0.3	1/14
γ Chlordane	0.04	ND - 0.5	1/14

ND=Not detectable

All the following pesticides were not detected (ND) in the 14 samples: VC-13, p,p'-DDE, p,p'-DDT, o,p'-DDE, β-BHC, HCB, Endrin, Trithion, Ethion, Ethyl Parathion, Methyl Parathion, Phorate, Methyl Bromophos, Fenthion, Chlorobenside, Phosdrin, Methoxychlor, and Toxaphene.

Table 30. PESTICIDE CONCENTRATIONS COLLECTED FROM A CLOTH SCREEN ($ng/\frac{1}{2}m^2$) FROM TWO SAMPLING SITES IN SOUTH FLORIDA. 1974

Pesticides	Cloth #1	Cloth #2	Cloth #3
VC-13	9.8	11.5	ND
Dursban	I	I	I
Diazinon	62.3	33.5	184.0
α BHC	I	5.1	3.4
Heptachlor	51.3	4.7	I
Lindane	22.2	20.4	18.1
Dieldrin	31.8	ND	ND
Ethyl Parathion	ND	ND	2,190.0
Methyl Parathion	ND	ND	325.0
Toxaphene	ND	ND	P
Sites	Suburban	Suburban	Allied Helicopter
Date of Collection	9/18	9/18-9/23*	9/23

ND=Not detectable
I=Interference
P=Present
*Five day collection sample
All the following pesticides were not detected (ND) in the 3 cloth samples: Malathion, Aldrin, p,p'-DDE, p,p'-DDT, o,p'-DDE, β- BHC, Endrin, Trithion, Ethion, Ronnel, Phorate, Methyl Bromophos, Fenthion, Chlorobenside, Phosdrin, Methoxychlor, αChlordane, γ Chlordane.

Table 31. TRAPPING EFFICIENCY OF NYLON SCREEN AND MRI IMPINGERS AT TWO SOUTH FLORIDA SITES. 1974

	Suburban			Allied Helicopter	
Pesticide	S #32 MRI Air Sampler (24 hrs) ng/m^3	Cloth #1 (24 hrs) ng/½m^2	Cloth #2 ng/½m^2	S #33 MRI Air Sampler (24 hrs) ng/m^3	Cloth #3 (24 hrs) ng/½m^2
VC-13	0.7	9.8	11.5	ND	ND
Dursban	1.4	I	I	1.6	I
α BHC	0.5	I	5.1	0.8	3.4
Heptachlor	1.2	51.3	4.7	I	I
Lindane	0.5	22.2	20.4	0.3	18.1
Dieldrin	1.0	31.8	ND	ND	I
Ethyl Parathion	ND	ND	ND	196.0	2,190.0
Methyl Parathion	ND	ND	ND	21.0	325.0
Phorate	ND	ND	ND	0.3	ND
Toxaphene	ND	ND	ND	P	P
m^3 Total Air Volume	49			40	
Date Collected	9/18	9/18	9/18-9/23	9/23	9/23

ND=Not detectable
I=Interference
S=Sample
P=Present

All the following pesticides were not detected (ND) in the two MRIs and the three cloth samples: Malathion, Aldrin, p,p'-DDE, p,p'-DDT, o,p'-DDE, β-BHC, HCB, Endrin, Trithion, Ethion, Ronnel, Methyl Bromophos, Fenthion, Chlorobenside, Phosdrin, Methoxychlor, α Chlordane, and γ Chlordane.

by the M.R.I. impinger, there was good qualitative concordance. Of particular interest to us was the ethyl parathion trapped by the screen which placed in the local environment of the helicopter site. As has been mentioned in the discussion of the urinary metabolite studies in this setting, we were surprised to see continuous excretion of parathion metabolites in the urine long after the completion of an occupational exposure as defined by pesticide application. The findings of pesticides in the ambient air could well be the reason why the metabolites were identified when there was no obvious work exposure.

For obvious reasons, the human and environmental aspects of dichlorvathion has been of special interest this last year. It was the regular identification of this pesticide in the air that prompted us to measure this pesticide after a single application. As will be seen from Table 32, the air concentrations were 0.54 ng/m^3 a week before the application, yet 10 days after application the air concentrations at the experimental site was 64.60 ng/m3, and it was 4.95 ng/m^3 23 days later and 1.3 ng/m^3 50 days after application! The environmental and human characteristics of this organophosphate pesticide identified during the last year certainly qualified this chemical as being persistent in both senses of the word.

Table 32. VC-13 CONCENTRATIONS (ng/m^3) IN AIR 10, 23 AND 50 DAYS AFTER A SINGLE LAWN SPRAY APPLICATION OF 26 OZ. OF 75% VC-13 SOLUTION

AIR CONCENTRATIONS			
Pre-Application	10 Days Post Application	23 Days Post Application	50 Days Post Application
0.54	64.60	4.95	1.30

SECTION VI

EEG STUDIES

EEG Studies - Background Information - Changes in the electroencephalograph following human pesticide exposure are dependent upon the type of pesticides involved and the nature of the exposure. In acute poisoning, the duration of cerebral anoxia has been shown to produce EEG changes. Hypothalamic spikes were noted by Holmes which persisted three years after parathion exposure. Brown reported persistent EEG changes similar to those seen in temporal lobe epilepsy following mild acute organophosphate exposure.[15] Metcalf and Holmes reported unusual EEG changes in organophosphate exposures.[16] Hunter and Robinson conducted EEG studies in three groups of volunteers fed 0.01, 0.05 and 0.211 mg of dieldrin per man per day, no abnormal EEG changes were noted with this amount of dieldrin intake.[17]

Animal studies recently conducted in the U.S. Environmental Protection Agency, Perrine Primate Laboratory by Dr. John Santolucito and others in the Pharmacologogical Branch have compared chronic and low level exposure effects of Carbaryl on the EEG of monkeys. Similar abnormalities have been noted following parathion, dieldrin and DDT exposure. Using a portable EEG machine suitable for battery operation in the field it was planned to conduct EEG studies in human volunteers who were occupationally exposed to pesticides.

Description - With the approval of the Human Experimentation Committee for the use of the portable EEG Unit whose only additional requirements were for an extra safety fuse within the circuitry to prevent any possible current overload. After a final electronic check, the field trials were initiated.

Field studies in pesticide exposed workers were conducted and volunteer acceptability was first observed. Thereafter EEG studies were conducted in the following categories: (1) multiple pesticide exposures, (2) single pesticide exposures. Tracings were collected in relation to the amount and degree of exposure to a variety of pesticide applications and those personnel involved in single pesticide exposure.

EEG tracings were collected from a single exposure to different chemicals. A tracing was obtained during a Baygon (Propoxur) application and upon completion when the applicator switched to Dursban, a second tracing was collected. EEG tracings were obtained during and after a VC-13 application. In addition, tracings were obtain after an acute intoxication with this pesticide.

After numerous consultations with Dr. John Santolucito, transference from the needle electrode to surface electrodes was adapted and EEG tracing were taken on one volunteer by the surface electrode first and immediately thereafter by the needle electrode technique.

Results - Tables 33a and 33b lists the demographic and exposure category of the 31 participants.

Problems Encountered - Volunteer acceptability was greatly enhanced with the switch to surface electrodes. Toward the end of the year reports were received of trouble with the playback unit in Research Triangle Park. Several corrective measures were taken but apparently with limited success. Until these in-house difficulties were resolved, Dr. Santolucito advised against further collection.

Table 33a. TABLE OF EEG PARTICIPANTS.

Identification	Age	Sex	Occupation	Classification of Pesticide Exposure	Categorical Type of Pesticide	Purpose of Recording	Type of Electrode
J.S.	39	M	Pilot Applicator	Chronic	Agricultural	Base Line	Needle
R.S.	55	M	Ground Applicator	Chronic	Parks and Recreation	Base Line	Needle
E.O.	45	M	Ground Applicator	Chronic	Parks and Recreation	Base Line	Needle
R.S.	55	M	Ground Applicator	Chronic	Parks and Recreation	Exposure to Sevin	Needle
E.O.	45	M	Ground Applicator	Chronic	Parks and Recreation	Exposure to Sevin	Needle
A.S.	54	F	Housewife	Ingestion	VC-13	Symptomatic	Needle
C.L.	45	M	Housing Unit Applicator	Chronic	Concentrated Dursban	Base Line	Needle
R.J.	42	M	Housing Unit Applicator	Chronic	Dursban	Base Line	Needle
J.P.	50	M	Formulating Plant Supvr.	Chronic	Multiple Exposure	Base Line	Needle
S.P.	22	M	Form. Plant Dispatcher	Chronic	Multiple Exposure	Base Line	Needle
F.B.	59	M	Form. Plant Loader	Chronic	Multiple Exposure	Base Line	Needle
A.P.	38	M	Formulator	Chronic	Multiple Exposure	Base Line	Needle
L.T.	21	M	Assistant Formulator	Chronic	Multiple Exposure	Base Line	Needle
A.S.	54	F	Housewife	Ingestion	VC-13	Asymptomatic Follow-up	Needle
J.D.	50	M	Researcher	Occasional	Volunteer Study VC-13	Pre-Study	Needle
J.P.	50	M	Formulating Plant Supvr.	Chronic	Multiple Compound	Low-Level Form. Activity	Needle
S.P.	22	M	Form. Plant Dispatcher	Chronic	Multiple Compound	Coordinated W/Air Sampler	Needle
F.B.	59	M	Loader	Chronic	Multiple Compound	Coordinated W/Air Sampler	Needle

Table 33b. TABLE OF EEG PARTICIPANTS.

Identification	Age	Sex	Occupation	Classification of Pesticide Exposure	Categorical Type of Pesticide	Purpose of Recording	Type of Electrode
A.P.	38	M	Formulator	Chronic	Multiple Compound	Coordinated W/Air Sampler	Needle
L.T.	21	M	Assistant Formulator	Chronic	Multiple Compound	Coordinated W/Air Sampler	Needle
J.D.	50	M	Researcher	Occasional	VC-13 Study	Post VC-13 Study	Needle) Simultaneous
J.D.	50	M	Researcher	Occasional	VC-13 Study	Post VC-13 Study	Surface)
R.D.	28	M	Agri-App. Pilot	Chronic	Multiple Compound	Seasonal Work Activity	Surface
R.S.	27	M	Agri-App. Pilot	Chronic	Multiple Compound	Seasonal Work Activity	Surface
G.J.	53	M	Agri-App. Pilot	Chronic	Multiple Compound	Seasonal Work Activity	Surface
B.I.	23	M	Agri-App. Mechanic	Chronic	New Employee	Control	Surface
A.C.	42	M	Agri-App. Pilot	Chronic	Multiple Compound	Seasonal Work Activity	Surface
L.S.	22	M	Agri-App. Mixer	Chronic	Multiple Compound	Seasonal Work Activity	Surface
T.J.	24	M	Agri-App. Mixer	Chronic	Multiple Compound	Seasonal Work Activity	Surface
R.S.	54	M	Ground App. Parks	Chronic	Multiple Compound	Azodrin	Needle
T.W.	19	M	Assistant Formulator	Chronic	Multiple Compound	Coordinated W/Air Sampler	Needle

SECTION VII

PERFORMANCE AND PROBLEMS ENCOUNTERED

Performance - Table 34 lists the number of chemical and biochemical analyses conducted during the study period and number of EEG tracings collected.

Problems Encountered - After injecting 144 samples of urine to determine the alkyl phosphates using the Shafik et al. method it was noticed at the end of August that the first fraction of the silica gel column chromatography which contains DMTP and DETP when concentrated to one milliliter and injecting five micrograms, negative peaks and sometimes ghost peaks appeared on the gas chromatograph using the flame photometric detector. The negative peaks interfered with the quantitation of these alkyl phosphates at ppb levels. The instrument was turned off and the inlet port, transfer line, head and base were subjected to ultra-sonic cleaning using different solvents. New columns were packed and after these had been cured and treated with carbowax and installed and in working condition, the problem still persisted.

These problems were discussed with Dr. T. M. Shafik in Research Triangle Park and he, after making some additional recommendations, agreed to investigate the problem in his own laboratory. He found that the original N-amyl N'-nitro-N-nitrosoguanidine precursor supplied by Aldrich Chemical Company had been packaged in glass containers. Their second production had been packaged in plastic containers which questioned the purity of the reagent and contained certain impurities. Aldrich Chemical Company was contacted and requested to investigate the reagent. At the time of the preparation of this report it appears that the problem has been resolved but we were unable to completely fulfill the alkyl phosphate requirements of the contract.

Table 34. TOTAL NUMBER OF ANALYSES COMPLETED DURING THE ANNUAL STUDY PERIOD.

Type of Analyses	Month and Number of Tests				Total Number
Urinary Alkyl Phosphates	Jan. - 16 Feb. - 31 Mar. - 24	Apr. - 32 May - 23 June - 18	July - 0 Aug. - 0 Sept. - 14	Oct. - 8 Nov. - 0 Dec. - 0	164
Urinary Phenols	Jan. - 23 Feb. - 12 Mar. - 39	Apr. - 13 May - 64 June - 11	July - 12 Aug. - 33 Sept. - 0	Oct. - 22 Nov. - 28 Dec. - 50	307
Blood Cholinesterases	Jan. - 71 Feb. - 4 Mar. - 24	Apr. - 14 May - 9 June - 51	July - 8 Aug. - 22 Sept. - 4	Oct. - 18 Nov. - 25 Dec. - 0	250
Air Samples	Jan. - 3 Feb. - 3 Mar. - 3	Apr. - 3 May - 3 June - 3	July - 5 Aug. - 5 Sept. - 5	Oct. - 3 Nov. - 3 Dec. - 2	41 + 12 blanks
Cloth Sample			Sept. - 1		1 + 1 spiked cloth + 1 blank
EEG Collected	Jan. - 1 Feb. - 2 Mar. - 2	Apr. - 1 May - 2 June - 5	July - 2 Aug. - 7 Sept. - 7	Oct. - 3 Nov. - 1 Dec. - 0	33

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GLOSSARY

EEG	- Electroencephalograph
N.D.	- Non-detectable
ppm	- Parts per million
ml/mOSM/l	- Milliliter per milliosmols per liter
Pl.	- Plasma
ChE	- Cholinesterase
n	- Number of persons
HUD	- Housing and Urban Development (Dade County)
Tr	- Trace
µg/ml	- Micrograms per milliliter
ng	- Nanograms
CFM	- Cubic feet per minute
m^3	- Cubic meter
PAM	- Protopam Chloride
RBC	- Red blood cells
ppb	- Parts per billion
vv	- Volume voided
DMP	- O,O-Dimethyl phosphate
DEP	- O,O-Diethyl phosphate
DETP	- O,O Diethyl phosphorothionate thiophosphoric acid
DMTP	- O,O-Dimethyl phosphorothionate thiophosphoric acid
Propoxur	- 2-Isopropoxyphenyl N-methycarbamate
Cygon®	- 0,0-Dimethyl S-(N-methylacetamide) phosphorodithioate-(Dimethoate)
Phosdrin®	- 0,0-Dimethyl 2 methoxycarbonyl-1-methyl vinyl phosphate
Dursban®	- 0,0-Diethyl 0-(3,5,6,-trichloro-2-pyridyl) phosphorodithioate
IPP	- 2-Iso-propoxyphenol

GLOSSARY

Baygon^{R}	- Aprocarb 2-isopropoxyphenly-N-methyl carbamate
DDVP^{R}	- Dichlorvos 2,2-dichlorvinyl dimethyl phosphate
Ronnel^{R}	- Fenchlorphos 0,0-Dimethyl (2,4,5-trichlorphenol phosphorothioate
VC-13	- Dichlofenthion (0-(2,4-dichlorophenyl) 0,0-dietyl phosphorothionate)
αBHC	- Alpha isomer of 1,2,3,4,5,6-hexacloro-cylcohexane
βBHC	- Lindane gamma isomer of 1,2,3,4,5,6-hexachloro-cyclo-hexane of 99+% purity

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